Manitoba Medical Review

Vol. 30 **APRIL, 1950** **No. 4**

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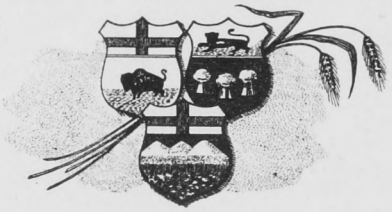

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NOTE:- To date there is no evidence of toxic reactions with Gravol. However, some individuals may become drowsy or confused on high or continuous dosage.

References:

1. Carliner, P.E., Radman, H.M., and Gay, L.H.: Science, 110: 215 (Aug. 26/49). 2. Gay, L. H., and Carliner, P.E.: The Prevention and Treatment of Motion Sickness. Bull, Johns Hopkins Hosp., May 1949. 3. Beeler, J.W., Tillisch, J.H., and Popp, W.C.: Proc. Staff Meet. Mayo Clinic (Sept. 14/49).

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Rutin	20 mg.
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DOSAGE

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The Manitoba Medical Review

Vol. 30 APRIL, 1949 No. 4

OBSTETRICS

Heart Disease and Pregnancy

L. R. Coke, M.D.

In "The Letter" in The Tale of Two Cities, Doctor Manette described a young woman, recently married, who had been the victim of two noblemen.

He said, "For an instant, and no more she would pause and listen, and then the piercing shrieks would begin again, and she would cry, my husband, my father, and my brother! and would count up to twelve and say, Hush! There was no variation in the order, or in the manner. There was no cessation, but the regular moment's pause, in the utterance of these sounds . . .

"I made the patient swallow, with great difficulty, and after many efforts, the dose that I desired to give. . . . I repeated the medicines I had given her, and I sat at the side of the bed until the night was far advanced. . . . She never abated the piercing quality of her shrieks, never stumbled in the distinctness or the order of her words. . . .

"I had come and gone twice, and was again sitting by her when she began to falter. I did what little could be done to assist that opportunity and by and by she sank into a lethargy. . . . It was as if the wind and rain lulled at last, after a fearful storm. I released her arms, and called the woman to assist me to compose her figure and the dress she had torn. It was then that I knew her condition to be that of one in whom the first expectations of being a mother have arisen; and then it was that I lost the little hope that I had had for her."

More of such literature might be quoted and through it popular attitudes towards pregnancy in those with uncertain health might be better understood. Fortunately it is not medical literature.

In 1906 French and Hicks reviewed the histories of three hundred women with Mitral Stenosis seen in the Outpatient Department of Guy's Hospital. Only ninety-five were unmarried The two hundred and five married women had an average of 4.5 children. One had seventeen children after the onset of her valvular disease. The authors had themselves admitted thirty-two of these patients and of these twenty-seven had heartfailure, before confinement. All of them survived labour. Five died at home within a period of three months, but autopsy showed that one death was due to endocarditis.

It was concluded from this study that there is no tendency to sterility or miscarriages in patients with Mitral Stenosis; that the treatment of heart-failure in Mitral Stenosis is the same whether or not the patient is pregnant, and that the likelihood that pregnancy will accelerate the time at which heart failure will develop is not as great as text-books suggest.

Speaking at University College Hospital in 1907 Blacker said, "I have chosen as the subject of this lecture Heart Disease Complicating Pregnancy and Labour," first because it is an important matter, and of not infrequent occurrence; secondly, because the teaching in some of the current text-books is, I venture to think, erroneous, in that it exaggerates the danger and the mortality arising from this complication of labour.

Blacker said that death in labour is rare. Death soon after labour is not uncommon. He described death in congestive failure which he associated with Mitral Stenosis and death due to shock, immediate or delayed which he found in patients with enlargement of the left ventricle and in those with a form of toxic myocarditis, in which post-mortem examination showed areas of fatty degeneration and haemorrhages in the heart muscle. In 453 cases, mostly rheumatic, his mortality rate within three months was 12%.

Blacker said: "The nature of the valvular lesion is not of much importance; it is the condition of the heart muscle that must be taken into account."

He induced abortion in cases of failure during the first months and induced labour prematurely in aortic valve disease as soon as the child was considered viable. He said, let them marry and he agreed with Hicks and French that they die whether or not they have babies.

Four years before his death Mackenzie published "Heart Disease and Pregnancy." He had retired from the London Hospital and had handed over other commitments to younger cardiologists. This book was not recommended by his biographer. A short summary follows: "Many women are subjected to unnecessary alarms and restrictions when pregnant; others have to suppress the natural desire of motherhood; while connubial relations are often disturbed by warnings and hints of danger that may never arise. . . . That the matter is not worse is in a measure due to the fact that the vast majority of women, especially in the working classes, never have their hearts examined. . . . The trend of modern medicine, both in teaching and in investigation, is to render the solution of

such problems as the relation of pregnancy and heart disease impossible. . . . The coming of the stethoscope was the beginning of a method in clinical research which has greatly hampered the practise of medicine, in that the introduction of mechanical methods has led to confusion as to the kind of knowledge which these methods are capable of affording. This criticism applies not only to the stethoscope . . . but to the polygraph, the electrocardiograph and blood-pressure instruments."

The difficulty in arriving at a diagnosis of heartfailure in the pregnant patient was described.

Heartfailure was defined as an inability of the heart muscle to maintain an efficient circulation. Important manifestations of failure were ischemic pain and breathlessness.

Oedema of the lower limbs was described as a feature of normal pregnancy and was thought to be related to changes in the veins. Crepitations in the lung bases were described as common in normal women during pregnancy, but were considered abnormal if they persisted after deep breathing or cough. Normal systolic murmurs, most commonly in the pulmonary area, but sometimes heard in other areas were described. Of these it was said, "The idea that a perfectly sound heart may show a murmur is so surprising to many doctors that they cannot accept it. Their view is that if a murmur is not organic it is functional and implies debility or some other condition of weakness. That a murmur may be as physiological as a pure sound is altogether outside the limits of their faith. . . . The detection of an innocent murmur has often supplied a reason for forbidding pregnancy or even marriage. Indeed when I ponder on this subject I am sometimes inclined to think that the discovery of the stethoscope has actually done more harm than good to the practise of medicine."

Of the pathological systolic murmurs it was stated that the loudest murmurs sometimes arose from a small opening in the ventricular septum, a condition that had little influence on the course of pregnancy. Accordingly, a systolic murmur, was to be considered unimportant unless there were other indications of heart disease.

"The detection of a mitral systolic murmur in a woman who is pregnant or who may become pregnant should cause the physician to consider the following points:

(1) The response to effort;

(2) The size of the heart;

(3) The rhythm of the heart.

In Mitral Stenosis when the heart is large or irritable, and when effort readily induces palpitation and breathlessness, then pregnancy should be forbidden. If . . . pregnancy has been undertaken, the case should be carefully watched, particular attention being paid to oedema of the lungs. If crepitations become persistent after cough or deep breathing, the advisability of inducing premature labour should be considered. If the percussion note of the lungs becomes impaired, interference is called for.

"My experience of aortic regurgitation complicated by pregnancy is limited to two cases. In these cases the heart was only moderately enlarged . Both made good recoveries, but were left very weak. Neither ever improved. They . . . died within two years.

"I . . . would allow pregnancy in a young woman with aortic regurgitation, if there was no 'Corrigan' pulse, if the heart was not enlarged or only slightly so, and if the response to effort was good. On the other hand, if there was a forcible apex beat outside the nipple line, a marked 'Corrigan' pulse and a distinct limitation of the response to effort, I should decide that pregnancy ought to be forbidden. Ectopic beats, often causing some discomfort, were described as occurring in about fifty per cent of healthy pregnant women. Paroxysmal tachycardias were not uncommon and treatment with digitalis was advised. (The writer of this paper has used quinidine, not knowing that the patient was pregnant and without incident, but recommends the established procedure of using digitalis). Some cases of auricular fibrillation, of short duration, and not associated with valvular disease were described and these patients withstood pregnancy without any difficulty."

The neurotic heart was described as "a distinct type of affection. The patients complain of pain in the left chest usually under the breast. The pain is of a dull aching character accompanied by stabs. . . . Attacks of pain may come on with great severity in the absence of any apparent provoking cause. These patients complain of exhaustion and the skin in the painful areas is sensitive."

"Pregnancy can safely be undertaken by these people. It often does them a great deal of good."

"The supposed danger of coitus to women with damaged hearts often mars the domestic happiness of married couples. . . . It is a matter for the woman, and not the doctor, to decide."

In congenital heart disease without cyanosis pregnancy was considered safe and two patients with patent ductus were described who survived more than one pregnancy without incident.

Hamilton of Boston has reviewed the present situation. He has proved that co-operation between obstetrician and cardiologist can save the lives of babies and mothers. In 1335 cardiac patients managed in the combined clinic the maternal mortality was 3.8%. It is reported that

the obstetricians who attend the Boston Lying-In are unable to diagnose mitral stenosis and must refer patients with murmurs or symptoms of heart disease.

Patients who have had failure, those that have auricular fibrillation and those that are in failure have their pregnancy terminated during the first three months. When failure develops later the patient is admitted to hospital and treated on ordinary lines for their heart trouble. During the last month of pregnancy the load becomes less. It is important to wait for this lightening. Delivery is by forceps from below as advocated by Mackenzie. In his favourable cases maternal mortality is 2.5%, foetal 12%. In his unfavourable cases the respective figures are 16% and 48%. Hamilton mentions the difficulty of making a diagnosis of essential hypertension. When it has been made the outlook in moderate hypertension is like that of favourable cardiac cases and that in severe hypertension, better than that in unfavourable cardiac cases.

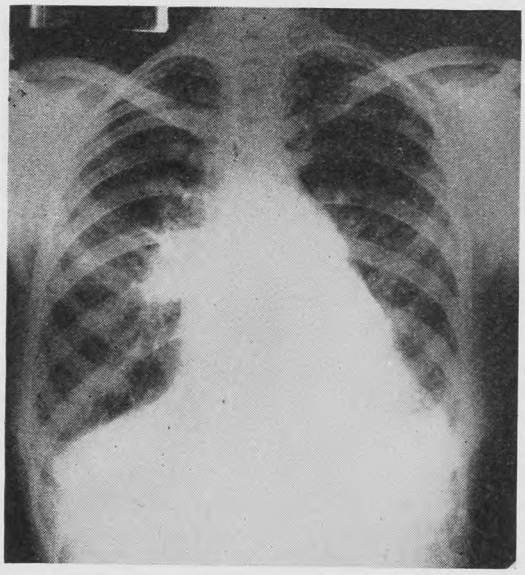
A new problem is that patients are now appearing with pregnancy out of the rheumatic age and into the degenerative age. The case of a woman who has had a baby after a myocardial infarct was described from the experience at the Philadelphia Lying-In Hospital.

This hospital reports on 409 cardiac patients, with a maternal mortality 3.4% and a death rate amongst babies of 13.2%. Sixteen pregnancies were interrupted during the first three months. Most of the other cases were delivered in labour from below. Caesarian operation was used where there were obstetrical indications and in some of the hypertensive patients. Some of the hypertensive patients had albuminurea, elevated B.U.N. and haematuria. It is their opinion that pregnancy does not alter the natural history of benign hypertension.

From Bellevue Hospital there is a report of 216 cases with interruption in eleven cases and a maternal mortality of 1.5%. Of the three deaths one was from endocarditis. Accurate measurements of the heart size as seen by radiograph showed that the patients with moderately enlarged hearts were not more likely to develop heartfailure than those whose hearts were normal in size. It was considered that the outlook was better if there had been a lapse of time between the attack of rheumatic fever and the pregnancy provided that this interval did not unduly increase the age of the patient. In this series there were six Caesarean operations done for obstetrical reasons. Thirty-six of their patients developed heart failure. It was considered that pregnancy, of itself, did not alter the natural history of rheumatic heart disease.

Some of the points mentioned in the literature are demonstrated in the case histories that follow.

Case No. 1. Born in England in the year 1900, the patient had her first episode of fever with painful swelling of the ankles and knees in 1913. Recurrences of polyarthritis appeared during the following winters, 1914, 1915 and 1916. In 1917 an examiner for the Q.A.I.M.N.S. discovered the presence of heart disease and her application for this service was rejected. After marrying a French-Canadian soldier in 1918, the patient came to Canada. In that year there was prolonged epistaxis associated with influenza. In 1920 the patient was kept in hospital for two months before the birth of her first child. In 1921 the second child was born without incident. In 1922 the third child was born. In 1923 digitalis was prescribed, which has been taken since. In 1924 the fourth child was born. Some oedema developed during the fifth pregnancy in 1926, but the patient and the infant survived. In 1928 severe haemoptysis



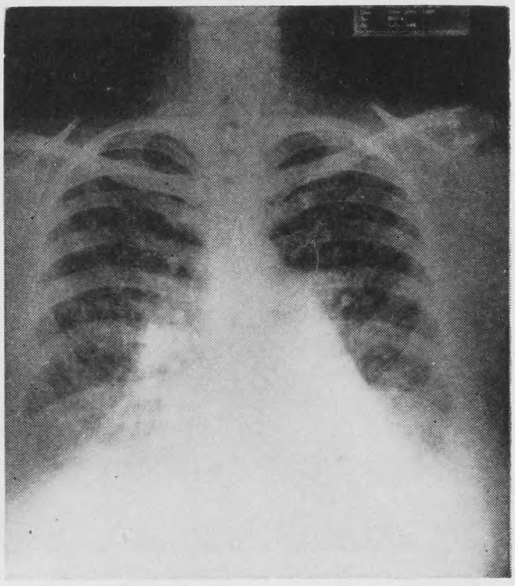
Case No. 1. X-Ray on the 15th Dec., 1949. Trans. Dia. of the Heart 15.5 cms.

required treatment in hospital. In 1934 the sixth child was born. In 1936 there was severe uterine bleeding after the birth of the seventh. In 1939 there was gross oedema during the eighth pregnancy, which disappeared after confinement.

A recent examination revealed a malar flush and auricular fibrillation. The cardiac apex was in the anterior axillary line. There was a loud systolic and a rumbling mid-diastolic murmur at the apex. The lungs were clear. The liver was firm and extended two and a half inches below the ribs. The blood pressure was 140/70.

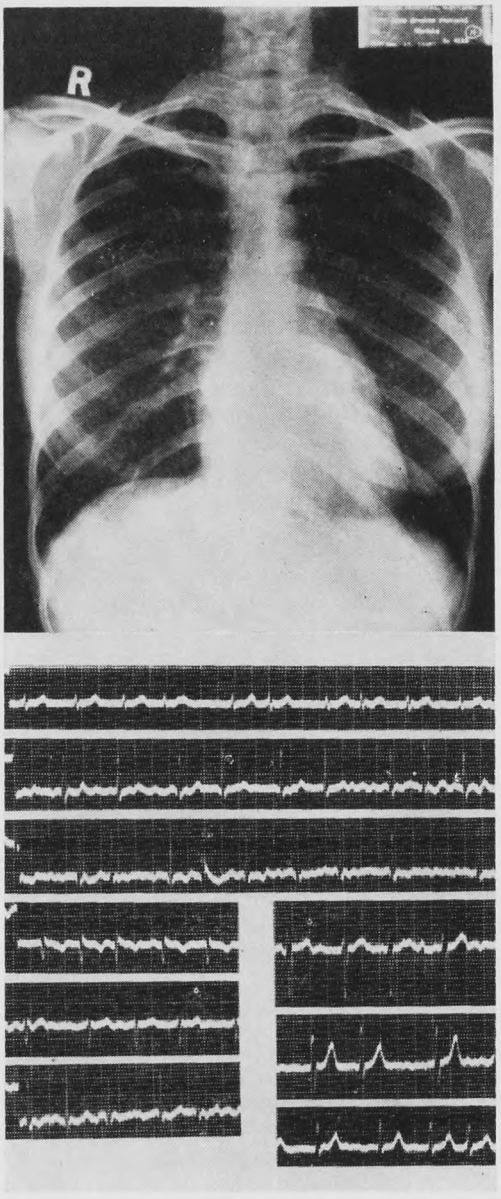
The patient is no longer able to do any work, but enjoys reasonable health and most of all her family.

Case No. 2. Born in 1909, the patient had chorea in 1924, before her marriage. Four children were born and her health remained good, but in 1932 polyarthritis and fever made it necessary for the patient to remain in bed for four months. From that date her health was indifferent. Examination in June, 1947, was as follows: the pulse was regular at 110, the cardiac apex 10.5 cms. from the mid-line and the mid-clavicular distance 8.5 cms. An apical systolic and a late rumbling diastolic murmur were present. The blood pressure was 120/70. There were crepitations in the lung bases and there was oedema of the ankles. The patient was believed to be at the beginning of the fourth month of pregnancy. Digitalis was precribed and when the signs of heartfailure disappeared a hysterotomy was done. In August, 1947, the patient was seen again. There was further enlargement and auricular fibrillation had developed.



Case No. 2. X-Ray on the 18th June, 1947. Trans. Dia. of the Heart 15.0 cms.

Case No. 3—Born in 1916, the patient spent most of the winter of 1928-29 in bed because of fever and joint pains. Subsequently treatment was prescribed for anemia and breathlessness restricted her activities. In October, 1949, breathlessness became more severe and the patient became unable to climb stairs. At the same time there was nausea and vomiting and amenorrhoea. Examination on the seventh of December, 1949, disclosed a slender young woman very short of breath with auricular fibrillation at a rapid rate, the apex 9.0 cms. from the mid-line and the mid-clavicular distance 7.5 cms., a loud first sound at the apex and a prolonged diastolic murmur, crepitations in the lung bases and a blood



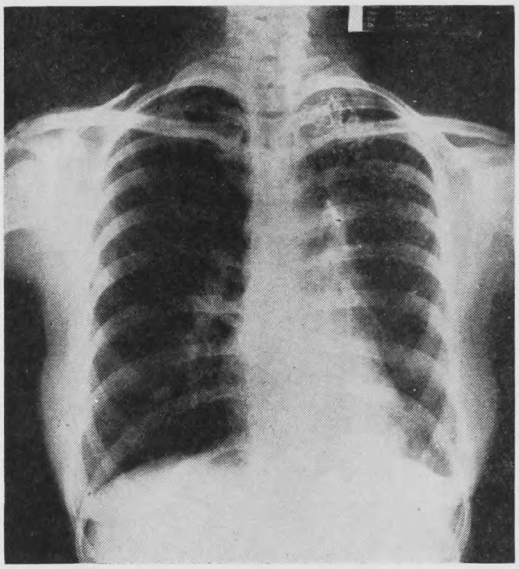
Case No. 3. X-Ray on the 10th Dec., 1949. Trans. Dia. of the Heart 14.0 cms.

Case No. 3. E.C.G. on the 8th Dec., 1949. Rate, 110; Rhythm, Auricular Fibrillation, Axis 90.

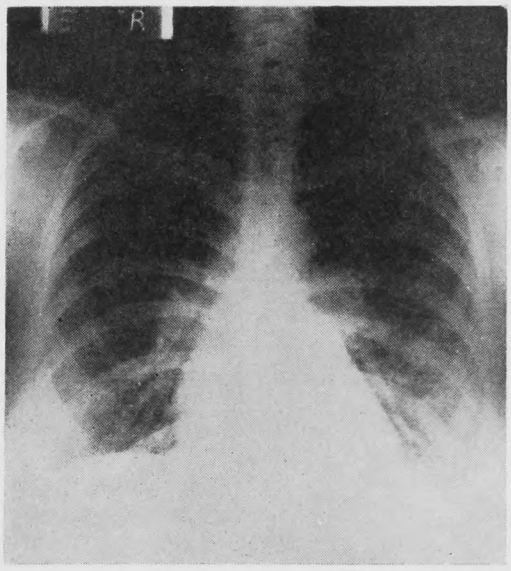
pressure of 115/65. The heart failure responded to digitalis, salt restriction and rest and hysterotomy was performed on December 17th.

Case No. 4. Born in 1918, the patient was confined to bed for three months in 1932 with fever and painful joints. In 1945, with weight loss and

tachycardia and a normal electrocardiograph, a thyroidectomy was performed. Following the operation the patient developed a left pleural



Case No. 4. X-Ray on the 10th May, 1949. Trans. Dia. of the Heart 13.0 cms.



Case No. 5. X-Ray on the 20th Nov., 1949, during the seventh month of pregnancy.

effusion and was kept in hospital for seven months. There was a fairly good recovery and the patient was married. In the sixth month of her first pregnancy, in 1947, breathlessness and oedema developed. Early in the eighth month a premature baby was born, but died, and the heart failure became so severe that continuous oxygen was required for ten days. In May, 1949, the patient was seen in the second month of pregnancy and interruption was advised.

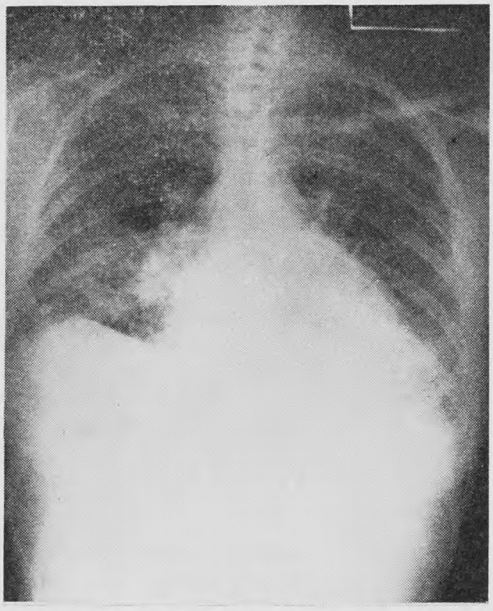
Case No. 5. Born in 1913, there was no history of joint pains nor chorea. The first three pregnancies were uneventful. In March, 1944, the patient had been admitted to hospital in the eighth month of pregnancy, when there had been complaints of breathlessness, vomiting, dizziness and specks before the eyes. At that time the heart had been considered enlarged and systolic and mitral diastolic murmurs had been recorded, also oedema of the lungs and lower limbs and blood pressure of 160/100.

Dititalis was given. Salt was restricted and labour was induced. Twin babies weighing five pounds three ounces and five pounds four ounces were delivered. The patient was advised against further pregnancies. In August, 1949, the patient was admitted to hospital very breathless, very frightened and four months pregnant. Her weight was 226 pounds. Pulse was 120, regular rhythm. The apex could not be felt, but X-ray revealed moderate enlargement. The mitral first sound was snapping in quality and systolic and diastolic murmurs were heard in the mitral area. There were no crepitations in the lungs. The blood pressure was 125/80, the haemoglobin was 58%. With reassurance and ferrous sulphate her symptoms subsided. Follow-up was unsatisfactory, but at term a seven-pound boy was born spontaneously, without medical attendance, and the patient was found to be in good health.

Case No. 6. A graduate nurse, born in 1909, had her first two children without any difficulty. Throughout her third pregnancy her attendance for prenatal examination had been regular and her blood pressure had been normal. Urinalyses and blood studies had also been satisfactory. March 10, 1948, her third pregnancy ended with the birth of twins. Labour had been of two hours duration. On March 20th the patient was discharged from hospital. On April 1st breathlessness was noticed. At the beginning breathlessness was more marked at night, but gradually the slightest effort became impossible, cough became troublesome and pink expectoration appeared. On the eighth of June the patient was readmitted to hospital complaining of: breathlessness, cough, faintness and palpitation. The neck veins were full and there was intense cyanosis. The apex was not felt but the apical rate was 112 and a gallop rhythm was noted in the apical region. The blood pressure was 98/70. There was dullness on the right side of the chest up to the second intercostal space anteriorly and there were crepitations in left base. X-ray revealed enlargement of the heart, especially of

the left ventricle, and showed fluid in the right pleural space.

With oxygen, morphine, salt restriction, digitalis and injections of salyrgan recovery was made. In August, in spite of careful restriction of activity, salt restriction and the daily use of Digitaline Nativelle O.1 milligrams, oedema of the lungs appeared with the onset of menstruation. This responded to a single injection of salyrgan. The patient has continued on restricted salt and Digitaline Nativelle and her exercise tolerance has gradually improved. In spite of some increase in weight, it is now possible for her to go up and down stairs and to do light housework.



Case No. 6. E.C.G. 4th May, 1948.
Rate, 110; Rhythm, Sinus, P:R: 0.15;
Axis, –30.
There is late inversion of T_1, T_2, Tf_4 and Tf_5.

Discussion

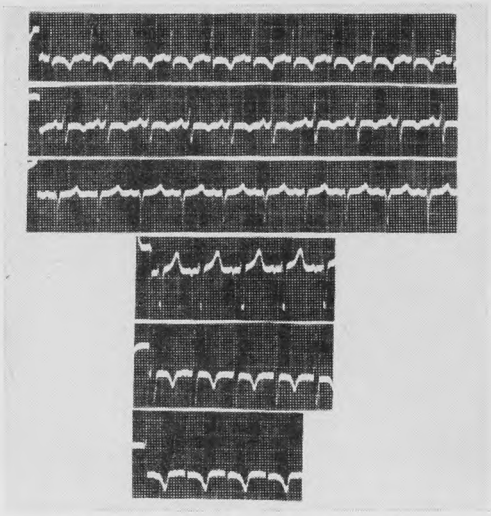
Case histories illustrating some of the features of heart disease and pregnancy have been given. These have included all the cases seen by the writer in which it has been considered necessary to interrupt pregnancy during the first months. The last case is considered one of Toxic Myocarditis.

Summary

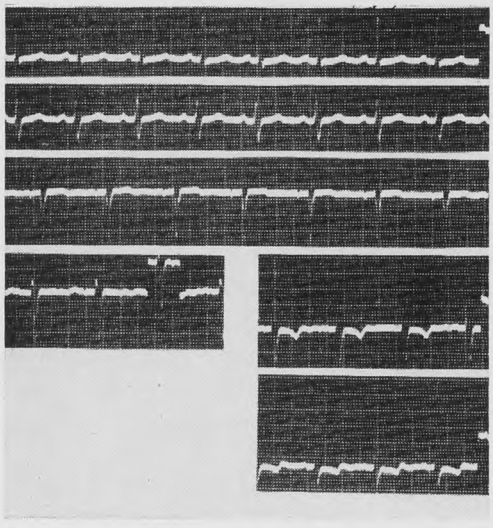
Gloomy forebodings about the heart in pregnancy are not based on observation or information. By and large the deepest gloom and the poorest information concerns the use of the blood pressure instrument, as predicted by Mackenzie. The other instruments have been of as little use and our course of action depends upon the presence or absence of heart failure.

Patients with heart disease have short lives. The natural history of their disease is not altered very much by their having some children. If heart failure develops in the first three months it is not due to the pregnancy, but the pregnancy should be interrupted.

If heart failure develops later the baby should be delivered from below at term.



Case No. 6. X Ray 9th June, 1948.
Trans. Dia. of the heart 15.5 cms.



Case No. 6. E.C.G. 10th Sept., 1948.
Rate, 78; Rhythm, Sinus, P:R: 0.16;
Axis, –30; T_1 and T_2 are now upright;
Tf_4 and Tf_5 are less deeply inverted.

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HAEMATOLOGY

On the Use of Unmatched Blood

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The use of so-called "universal donor" blood for transfusions, emergency or otherwise, has provoked controversy since its introduction by Ottenberg[1] in 1911. The position was complicated by the discovery of the Rh factor and the literature has now grown to formidable proportions. The majority of physicians and surgeons find it a matter of extreme difficulty to keep abreast of subjects outside their own field and we therefore believe it would be of value to summarize the present state of opinion on the use of "universal donor" blood. We will also put forward certain suggestions, the adoption of which would materially assist a steady supply of blood from any blood bank.

The Use of "Universal Donor" Blood

The small series of cases described by Ottenberg consisted of patients of Groups A, B or AB transfused with Group O blood. There were no reactions in the series and this was explained as being due to dilution of the anti-A and anti-B agglutinins of the donor blood by the large bulk of the patient's plasma. With the increasing use of "universal donors," however, reports of hemolytic reactions, some fatal, began to appear and usually concerned donor blood in which the iso-agglutinins were of an unusually high titre. If freedom from reaction depended purely upon dilution of the transfused iso-agglutinins, it is obvious the likelihood of such a reaction would increase with the volume transfused as well as with the iso-agglutinin titre. Modern studies, such as that by Aubert, Boorman, Dodds and Loutit[2], indicate that infusion of a serum with high agglutinin titre more often gives rise to a destruction of the recipient's red cells spread over days or weeks and detectable only by laboratory methods than to frank clinical reactions. While such a silent elimination of transfused cells detracts from the value of the transfusion, this is not a matter of any great importance if "universal donor" blood is reserved for unmatched use in cases of grave emergency only, where the essential indication is for restoration of blood volume rather than of red cell count.

Quite apart from the dilution factor postulated by Ottenberg, it has been shown by Levinson and Cronheim[3] that agglutinogen present in the patient's serum will, to a greater or lesser extent, neutralize transfused incompatible agglutinins and thus the action of the latter on the patient's red cells is prevented. It is apparent that the possibility of a reaction depends upon several factors, including the volume transfused, the agglutinin titre, and the amount of free agglutinogen in the recipient's serum. It is not surprising, therefore, that there is a wide divergence of opinion as to the precise agglutinin titre which should be considered dangerous and this also varies with the technique used in performing the titration, as the results from different techniques are not comparable. Morgan and Lamb[4] suggested an extreme of 1:256, while Aubert et al recommended avoidance of titres of 1:512, and the most recent article on this subject (Ervin and Young[5]) mention it to be common practice to consider titres below 1:200 as "safe." The last authorities comment upon the lack of agreement on this rather important matter.

From the extensive literature, only a few articles of which have been quoted, it is obvious that the practice of giving "universal donor" blood is widespread and that it is so indicates that the associated risks must be small. Solandt[6] operated a blood depot in which about 900 whole blood transfusions were given each month to air raid casualties, almost all the blood being of "universal donor" group and given without cross-matching. If hemolytic reactions had been frequent the policy could scarcely have been maintained. Levine and Maybee[7] in 1923 emphasized the dangers of high titred Group O blood, but by a subsequent communication in 1945, Levine[8] expressed the opinion that the dangers had been stressed unduly.

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However much disagreement there may be upon what constitutes a dangerous level of agglutinin, it can be taken as agreed that unduly high titres should be avoided and this may be done in one of two ways. Firstly, all Group O blood may be subjected to a single examination at a predetermined dilution; should agglutination occur, the blood is not used unmatched. Secondly, the iso-agglutinin may be neutralized by group specific substances, as first suggested by Witebsky, Klendshoj and Swanson[9]. These authors demonstrated that such neutralization is effective, but there is one objection, possibly of more than academic importance, to such a procedure. Tisdall, Garland and Wiener[10] pointed out that group specific substances are in themselves antigenic, even when combined with corresponding agglutinins, and thus they may provoke an immune response. The possible significance of this is where the patient so transfused is a female who subsequently carries a child of an incompatible ABO group. Whilst hemolytic disease of the newborn due to A-B sensitization is less common than when due to Rh incompatibility, Wiener, Wexler and Hurst[11] suggest that such disease in an A-B incompatible pregnancy is particularly liable to occur when iso-agglutinins of an immune type are present and such antibodies are of the type which would be produced by injection of group specific substances. On the whole, therefore, it would seem that the selection of Group O blood with low or moderate titres of iso-agglutinin is preferable. That such a policy would result in adequate supplies of "universal donor" blood for emergency purposes is shown by the fact that an accepted end-point of 1:128 embraces approximately 75% of all Group O donors.

The policy of several large transfusion services, including that operated by the Canadian Red Cross Society, is to make available Group O blood for use unmatched in cases of utmost emergency where the delay of even only three or four hours before cross-matched blood could be provided would prejudice survival of the patient. On such a basis it is apparent that the slight risk involved with the use of "universal donor" blood, even if the iso-agglutinin titre has not been determined, is quite outweighed by the urgency of the case. It must also be remembered that, in such cases, the indication is for restoration of blood volume rather than of red cell count and thus plasma can also be used to cover the period before cross-matched blood is available (Levinson, Rubovits and Necheles[12]). When the blood bank adopts the policy of excluding, for emergency purposes, Group O blood with a high agglutinin titre, the safety of the procedure is increased. In the Manitoba Depot it is the policy to exclude for this purpose any blood which shows a definite agglutination at a titre of 1:128.

The Question of the Rh Factor

It is a natural tendency that, to a patient whose Rh type is unknown, Rh negative blood is given in case of emergency. Under the previously mentioned policy of several large scale transfusion services, such a practice has one serious drawback which should be appreciated by all responsible for ordering emergency transfusions. In round figures, in a mixed white population slightly less than 50% will be found to be of Group O, and of these, only about 15% are Rh negative. In other words, from a hundred random donors only seven or eight Group O, Rh negative bottles of blood can be expected and this small store has to serve, not only as cross-matched blood for Group O, Rh negative recipients, but also for unmatched emergency transfusions. The problem is greatest in the first six months of the operation of a blood depot because, after that period a large panel of donors whose group and types are known is available. It is also obvious that about 85% of all the cases to whom emergency blood is given will be Rh positive. Now, if Group O, Rh negative blood could be made freely available and if the administration to the Rh positive recipient were free from danger, there would be no difficulty. However, the supply of Group O, Rh negative blood will always be a major problem for any transfusion service and, moreover, as Diamond[13] points out, Rh negative blood given to Rh positive patients may result in the production of antibodies to the "Rh negative" factors. With these facts in mind, and knowing that Rh antibodies have never been found to occur naturally, the question of administering Rh positive blood, unmatched, to patients whose type is not known must be faced. On a statistical basis the chances are 85 to 15 that the patient will be Rh positive, but this fact alone, encouraging as it may be, is not enough. If Rh positive blood always produced immunization of the Rh negative individual and if hemolytic reactions always followed one such transfusion, then the policy would be untenable. However, experience of the war years indicates that less than 50% of the Rh negative war casualties, transfused regardless of the Rh factor, became immunized and this despite the fact that many, if not the majority of these individuals, had received multiple transfusions (Diamond cited by Stanbury[14]). While Diamond observed little differences in the percentage sensitized of those with single and those with multiple transfusions, Mollison, Mourant and Race[15] point out that it usually requires multiple transfusions to produce significant titres of Rh antibodies and, in such cases, the development of antibodies is gradual, early reactions being mild and such "warning" symptoms being adequate to draw attention to the necessity for full grouping, typing and cross-matching should this not have been performed previously.

The discrepancy between the two opinions is probably due to the fact that the immunological effect of multiple ransfusions is the same as that of a single injection if the former are given in rapid succession, i.e., within a few days. Bearing in mind that we are discussing the administration of "universal donor" blood to cases of critical urgency, then it is obvious that the likelihood of any one patient requiring more than one such transfusion during his life is remote.

The administration of Rh. positive blood to an Rh negative recipient carries danger only in certain types of cases. Above all, such a procedure may result in sensitization of an Rh negative girl or woman, and Diamond is only one of several authorities who have found that the most severe cases of erythroblastosis are encountered in children of women who have been sensitized by previous transfusions. Levine and Waller[16] showed that even an intramuscular injection of Rh positive blood in infancy or childhood may sensitize an Rh negative female, with the appearance of erythroblastosis in her first born. A second group of Rh negative patients to whom Rh positive blood should not be given are women past the child-bearing years of life whose obstetrical history suggests immunization to the Rh factor. Lastly, any patient who has had previous transfusions may have been sensitized and thus should receive Rh negative blood in an emergency.

Conclusions

From this discussion a suggested policy emerges. In cases requiring immediate transfusions (that is, where the delay necessary for cross-matching would endanger life) Group O blood of low or moderate agglutinin titre may be used with negligible risk. Due to the fact that Rh antibodies have never been found to occur naturally, and that the Rh factor is of comparatively inconstant and weak antigenicity, Rh positive blood may be given with considerable safety to all patients requiring emergency transfusions with the exception of four definite categories:

(1) Girls and all women in the child-bearing years of life.

(2) Women past the menopause who have an obstetrical history suggesting immunization to the Rh factor.

(3) All patients with a history of previous transfusions.

(4) Comatose patients and others unable to give a clear history.

It also follows from all that has been said on the Rh factor that Rh positive blood may deliberately be given to Rh negative patients provided the latter have not previously been sensitized. This policy can be advocated especially in those who suffer an incurable disease such as leukemia, carcinoma and true aplastic anaemia. Naturally a percentage of such patients will develop antibodies, but these will be detected by the routine methods of cross-matching before harmful effects become apparent. In the majority it is obvious that immunization will never occur, either because of natural inability to react to the antigen or because of the intervention of death. Such a practice would spare a considerable quantity of Rh negative blood for those patients who would benefit most by it.

Summary

A discussion is presented on the unmatched use of "universal donor" blood in grave emergency and on the selection of the Rh type of such blood.

It is suggested that Rh positive blood be used deliberately in transfusions for certain classes of Rh negative patients.

It is emphasized that the maintenance of adequate supplies of Group O, Rh negative blood in any blood bank is a problem which could be eased considerably by careful consideration of the suggestions presented in this article.

Acknowledgements

While the opinions expressed in this article are personal and do not necessarily reflect the official policy of the Canadian Red Cross Society, we are indebted to Dr. W. Stuart Stanbury, National Director of the Blood Transfusion Service, for constructive criticism and for constant encouragement. The value of the article has been enhanced by the incorporation of suggestions from Dr. Bruce Chown.

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PAEDIATRICS

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Cystic Fibrosis of the Pancreas

Evelyn Alice Loadman, M.D.

An analysis of the cases in the Children's Hospital, Winnipeg, 1945-1949.

During the period from 1945 to 1949 there were 22 cases of Cystic Fibrosis of the Pancreas on record in the hospital cross-index. The records of these cases have been studied, and an attempt was made in every case to follow the child and discover the ultimate outcome of the illness.

As a result of these studies, the following analysis of these cases was made.

Group I

Three of these 22 cases were discarded—they defied analysis because there was insufficient investigation carried out to make a satisfactory diagnosis, and follow-up investigation proved impossible.

There were six cases which, it was felt, were proved to be Cystic Fibrosis—two were proved definitely by autopsy, and four by clinical investigation and follow-up. Five of these six cases died, although there were only two upon whom autopsy was performed, and the remaining one is under supervision and modern therapy.

There were nine cases which, it was felt, were proved not to be Cystic Fibrosis, one by autopsy and eight by follow-up. Of these eight children, who were followed, five showed no further evidence of the disease after hospitalization, and in three, the diagnosis was established along other lines.

The four doubtful cases at the bottom of Figure 1 might well be included in the "proved not to be" class, bringing the total to thirteen.

Thus in the nineteen cases in which some follow-up investigation was possible, six cases were proved definitely or most likely to be Cystic Fibrosis, and thirteen cases were proved definitely or most likely not to be Cystic Fibrosis.

It will be realized by now that there is a strong element of personal opinion in this analysis and it is necessary, therefore, to state the criteria used in order to make the diagnosis. Significant autopsy findings are, of course, definite proof of the disease. Clinically the diagnosis was accepted in this particular series only on the following grounds.

1. The child must be ill with evidence of a serious disease.

2. The clinical and laboratory findings must be full-blown. With our present diagnostic armamentarium it is not felt that mild or subclinical types can be diagnosed.

3. The disease must show evidence of chronicity or prove fatal.

What are the clinical findings which may be accepted as suggestive of Cystic Fibrosis? This information is available in most Paediatric textbooks, but a brief review may clarify the discussion to follow.

First—A positive family history is suggestive. The disease is thought to be a Mendelian recessive hereditary characteristic. In Zeulzer's series, out of 21 families with siblings, 7 cases had a positive family history.

It is a disease process which begins just before, at, or shortly after birth. There is a rapid progression of the disease and symptoms are produced promptly. According to the literature 80% of cases have symptoms before three months of age.

Second—Intestinal Symptoms—These may be divided into two types of cases.

1. Those with Meconium Ileus. Cystic Fibrosis is now considered to be the foremost cause of this disease. The disease process affects the intestinal glands and secretions, with the production of a dark, tenacious, rubbery meconium.

2. Those with evidence of pancreatic insufficiency. Great variation in symptoms is produced by pancreatic insufficiency because the child often has a ravenous appetite which compensates to a certain extent for the loss of foodstuffs due to poor digestion. Frequent and/or foul stools and abdominal distention are the only gastrointestinal findings of any significance. Steatorrhea is often observed as a late finding, but it must be remembered that excess fat may not be evident from clinical examination of the stool, and may be present earlier than we think.

The laboratory tests which aid in the diagnosis of pancreatic insufficiency are:

1. Trypsin assay of stools and duodenal juice. Stool examinations for Trypsin have been done at this hospital for only six months and the value of this test remains to be proved in our own experience. Examinations of the duodenal juice for Trypsin have been done here since 1943 and great reliance has been placed on this test. From observations made on these cases, it seems that, in itself, the discovery of absent trypsin is not conclusive proof of the disease. More will be said about this later.

2. Steatorrhea, Azatorrhea and Amylorrhea are all suggestive findings but not proof of the disease.

3. Vitamin A tolerance test—this again is a helpful test but decreased absorption of Vitamin A is not diagnostic.

4. Glucose tolerance curve—is often flat in Cystic fibrosis but other types of curves may be shown.

5. Clumping of the barium meal in X-rays of the small bowel is sometimes shown, but this is also demonstrated by some celiac patients.

6. Gelatin tolerance test—this is not yet in use here, but it seems to be a logical method for testing tryptic activity within the body.

Third—Pulmonary symptoms. According to the literature and to our findings here there are few, if any, cases without pulmonary lesions. Clinically the typical symptoms are early onset of cough and dyspnea—the latter may be progressive and lead to some degree of cyanosis. Coarse sticky rales and emphysema are the commonest findings. The pulmonary lesions often determine the course of the disease—the patient may die of anoxia with little sign of infection, or widespread staphylococcal pneumonia may be the cause of death. More chronic cases result in bronchiectasis.

Fourth—The nutritional state. This is never normal. Typically there is a failure to gain and grow on an adequate diet. The appetite is excellent to ravenous. Evidence of rickets is absent, because the child does not grow, although it might well be expected, due to poor absorption of the fat soluble vitamins. Osteoporosis, however, is a common finding.

Fifth—Cardiac lesions may develop secondary to the pulmonary lesions. Some cases in the literature—have been described as showing rapid cardiac enlargement with failure, i.e., a cor pulmonale. We have not observed this here.

Sixth—The clinical course. Modern treatment modifies our conception of the disease as being fatal in all cases. Nevertheless on the basis of the pathology present, Cystic fibrosis should not be diagnosed, it is believed, unless there is a chronic course. In this series, therefore, the cases which showed no further evidence of pulmonary or gastrointestinal symptoms following hospitalization, were not diagnosed as Cystic Fibrosis.

Figures II, III, IV give a further analysis of the nineteen cases in which it was possible to obtain some follow-up investigation.

Group II

This group of six cases are the ones which were proved by autopsy (marked X or by clinical findings to be reasonably definite cases of cystic fibrosis of the pancreas. The first five columns represent the clinical findings, suggestive of the disease—family history—respiratory symptoms (of cough, dyspnea, cyanosis, positive X-ray findings) —abnormal stools—abnormally good appetite—abnormally poor nutrition.

As shown in this figure, the clinical findings are, for the most part, positive—marked yes—that is, in favor of the disease. Three of these had a positive family history. Respiratory symptoms were marked, harassing cough beginning in all cases before 7 months. The fatal cases all died a respiratory death with cyanosis intervening sooner or later. Broncho pneumonia was diagnosed at one time or another in all cases. Frequent or foul stools were present in four of these children this condition beginning early in life and was recurrent. One had a history of a meconium plug at birth. The appetite in all cases was good, sometimes abnormally so, yet the nutrition was poor. Trypsin was markedly reduced or absent. The clinical course was fatal in all but one and this patient is under treatment with aureomycin and pancreatin and is doing remarkably well.

In this group, therefore, the diagnosis seems reasonably certain. The overall clinical picture is one of positive findings in favor of the disease. Trypsin was reduced or absent. All but one had a serious fatal disease. Unfortunately, none of these cases demonstrate the chronicity of the disease, as the longest history was of nine months duration, and the histories of the remainder were relatively short. The longest period over which any of these children were observed in hospital was three months.

Group III

This figure represents the cases in which trypsin was again reduced to very small amounts or absent —but in spite of this significant finding, the diagnosis was disproved by follow-up investigation. These cases are divided into two groups because the first group showed few symptoms typical of the disease and the second group presented a fairly characteristic picture.

In the first group some children had one or two attacks of diarrhea, or one attack of bronchopneumonia, but there was no intermittent persistence of symptoms. The appetite in this group was poor. These children were all malnourished, but this is not a very diagnostic finding. Furthermore, these children were all well soon after discharge, with no recurrence of symptoms. One was mentally retarded, but physically well.

The absence of trypsin in the duodenal juice in these five cases which seem not to be cystic fibrosis either clinically or by follow up, is not readily explained. The position of the tube in the duodenum was checked by fluoroscopy in all cases, and the pH was tested. However, the duodenal juice was obtained in all cases after the child had been fasting for six or more hours, and no pancreatic stimulant was used. The normal stimulation which sets into motion the mechanism leading to pancreatic activity, is the passage of acid chyme into the duodenum. It seems reasonable, that abnormally low or absent trypsin might be found in the duodenal juice in any sick child in the fasting state, when no attempt is made to stimulate

the pancreas. Complete gastric achylia is not diagnosed without histamine and it seems logical to use a pancreatic stimulant when testing for pancreatic achylia. (Recent work suggests that secretin stimulates a flow of pancreatic juice low in enzymes and is therefore not the stimulant of choice). The failure to use a pancreatic stimulant, may therefore be the explanation for the absence of trypsin in these cases.

In Figure III the lower group of four cases is separated from the upper group, because here the clinical findings were much more suggestive. There are two with suggestive family histories. Trypsin was again absent or markedly reduced in all cases. While these children were in hospital the diagnosis was on firm grounds. However, after leaving the hospital, S. B. died of tuberculosis, which was suspected but not proven during her hospitalization, D. M. and J. R. made good progress in all respects with no symptoms suggestive of the disease, and the fourth, K.K., died, and autopsy failed to show evidence of Cystic Fibrosis. These four are problems—clinically at the time of their hospitalization, who could dispute the diagnosis? However, if trypsin had been found in normal amounts in the duodenal juice—and the suggestion is made that it might have so been found, if a pancreatic stimulant had been used, the diagnosis might never have been made.

Group IV

In case it may be thought from what has been said, that this test for trypsin is worthless, let us examine this last group of cases which also were proved not to be Cystic Fibrosis. The diagnosis was made, in spite of the fact that the trypsin assay was normal, because the remaining clinical findings were so suggestive (marked "yes"). In none of these cases was the diagnosis confirmed by follow-up investigation. E.P. is now known to be a victim of congenital heart disease with pulmonary infection. S.W. undoubtedly had bulbar poliomyelitis and secondary pulmonary infection from which she has recovered. W.N. had an uneventful recovery following hospitalization with no recurrence of symptoms. S.C. died and the autopsy findings were marasmus only.

There is little scientific information to offer as a result of the study of these cases. They have all been studied in retrospect, which is the most comfortable way in which to make a diagnosis of Cystic Fibrosis. Studies included an analysis of the patients' charts and follow-up investigations which varied from a minimum of 6 months to maximum of 5 years. Nothing has been proved, for the analysis of these cases is mostly an expression of personal opinion. However, a few points have arisen which may warrant consideration.

Firstly—If trypsin is present in normal amounts in the duodenal juice, the diagnosis may be ruled out. If it is absent, it cannot be assumed that the diagnosis is made on this finding alone.

Secondly—When testing for pancreatic achylia, it seems logical to use a pancreatic stimulant.

Thirdly—The gelatin tolerance test seems a more logical and more informative test for trypsin activity than any other so far devised, and its diagnostic value should be investigated by further trial in suspected cases.

Fourthly—Although all evidence points in favor of a diagnosis of Cystic Fibrosis, it is wise to withhold final judgment until a child is studied for a considerable period.

Group I

Cases on record	22		
Cases discarded—insufficient investigation	−3	=	19
Cases proved to be—by autopsy	2		
—by follow up	4		
	—		6
Cases proved not to be—by autopsy	1		
—by follow up	8		
	—		9
Cases doubtful			4

Group II
Cases proved by autopsy (or practically certain clinically)

Family History	Resp. Symp.	Abn. Stools	Good Appetite	Poor Nutr.	Trypsin Assay	Follow Up
S.C. yes	yes	no	yes	yes	.....	died
J.C. yes	yes	no	yes	yes	low	died
C.B. no	yes	yes	yes	yes	absent	died
K.M. no	yes	yes	yes	yes	absent	died
G.S. no	yes	yes	yes	yes	absent	died
K.P. yes	yes	yes	yes	yes	absent	OK

Group III
Cases proved not to be or doubtful (trypsin reduced or absent)

Family History	Resp. Symp.	Abn. Stools	Good Appetite	Poor Nutr.	Trypsin Assay	Follow Up
J.Mc no	no	twice	no	yes	low	OK
D.S. no	no	no	no	yes	absent	OK
F.S. no	once	once	no	yes	low	OK
H.R. no	no	once	no	no	absent	OK
F.M. no	no	once	no	yes	low	OK mental
S.B. no	late	late	yes	yes	absent	Tb
D.M. no	yes	yes	no	yes	absent	OK
J.R. yes	yes	yes	yes	yes	absent	OK
K.K. yes	yes	yes	no	yes	low	died X

Group IV
Cases proved not to be (trypsin normal)

Family History	Resp. Symp.	Abn. Stools	Good Appetite	Poor Nutr.	Trypsin Assay	Follow Up
E.P. yes	yes	no	yes	yes	normal	Cong. heart
W.N. no	yes	yes	yes	yes	normal	OK
S.W. no	yes	no	yes	yes	normal	B. polio
S.C. no	yes	yes	no	yes	normal	died X marasmus

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OTOLARYNGOLOGY

The Problem of Deafness

Maurice M. Pierce, B.A., M.D.

The problem of deafness is a serious one. All of us have, at one time or another been confronted with this problem. Statistics suggest that approximately 10% of the population suffers from some form of hearing disability.

We usually classify the deafness as either:

(1) Conduction deafness.
(2) Perception deafness.
(3) Oto sclerosis.

One cannot be too dogmatic in any classification of disease and this also holds true in deafness; because frequently there is no "pure" conduction deafness or "pure" perception deafness, but a mixture of the two.

The Conduction type of deafness or middle ear deafness, is one which we all see very often. This type of deafness is the result of obstruction to the passage of sound vibrations to the inner ear (that is, to the cochlea and auditory nerve). The pathology may be present anywhere in the external canal, middle ear or Eustachian tubes; for example, wax in the canal; otitis media; Eustachian tube obstruction, etc. The treatment here is directed to the cause and this type of deafness is as a rule amenable to proper treatment.

The Perception type of deafness or nerve deafness, is the result of pathology which interferes with the cochlea or the pathways of the auditory nerve. This type is due to Labyrinthine infection; Toxemia; Drug poisoning, e.g. quinine, salycilates, streptomycine; Central Nervous System diseases such as Disseminated Sclerosis; Syphilis; 8th nerve tumors, etc.

Oto sclerosis is the other major group in the above classifications of deafness and it is this group that I wish to discuss in this paper and it is the diagnosis of this type of deafness which I wish to emphasize. The reason for this being that, until recent years, the diagnosis of otosclerosis categorized the patient's deafness as hopeless, however, as I will outline later, that "hopeless" prognosis is no longer tenable.

Otosclerosis is a term applied to a progressive bilateral loss of hearing, involving first the conduction apparatus and in the later stages the perceptive mechanism.

It becomes apparent in early adult life, beginning after puberty; is commoner in female than in males and is frequently associated with tinnitus. The onset is as a rule insidious with a low tone loss, with or without tinnitus; however, it may become "malignant" and progress to total deafness very rapidly.

The etiology is uncertain but hereditary predisposition is considered one of the basic factors. Pregnancy may precipitate it and on occasions aggravate it.

The pathology of "clinical otosclerosis" consists of a developmental proliferation of new, vascular spongy bone involving the area of the footplate of the stapes and the oval window. The most frequent site or focus of election is near or in a bony fissure (fissure ante fenestrum) which is located just anterior to the oval window. **This invasion of new bone causes a fixation of the stapes and thus interferes with the conduction of sound to the cochlea.**

You will note that in the above paragraph I said "clinical otosclerosis." However, otosclerotic foci may be found anywhere in the bony capsule of the labyrinth and it is often found at autopsy in people who have never been deaf or known to be deaf. However, to cause "clinical otosclerosis" it must involve the area of the footplate of the stapes resulting in its fixation and thus the prevention of proper sound conduction.

On otological examination of such an individual no pathology is discernible. The external canal is clear and occasionally is remarkably free from wax; the drum is normal in appearance and the Eustachian tubes are patent.

In an early case the tuning fork tests will reveal a conduction type of deafness with an Air Conduction loss (negative Rinne) and a lateralization of the sound to the affected side.

The audiometric examination is also typical of a low tone loss especially in the 128 to 1024 D.V. frequencies. The loss will range from 25 to 50 decibels and down.

In the later stages it will also show a high tone loss which will approximate the low tone loss giving a typical flat otosclerotic curve.

The bone conduction as recorded on the audiometer will in the early cases show a normal curve with no loss; in the later stages it will show the associated perception loss.

The 25-30 decibel level is considered the lowest range for serviceable hearing; a drop below this level, especially in a 512 to 2048 D.V. frequencies results in the loss of serviceable hearing.

Audiometric examination is important; not that the findings are more accurate than the findings with a careful fork examination; but that one also has a graphic representation of the loss for further comparison.

The subjective findings must not be overlooked in our attempts at making a diagnosis. These findings are common in conductive deafness. The individual is frequently "soft-spoken" because he

is not too aware of external noise and he does hear his own voice loudly. He will also frequently tell you that he hears better in a noisy environment—this is usually due to a fact that people will speak louder to make themselves heard in such an environment and too, the deaf individual is not as aware of extraneous noises. I wish therefore, at this point, to again emphasize the following:

"That any progressive bilateral hearing impairment of a conductive type, occurring in early or middle adult life, which on clinical examination does not show any Ear, Nose or Throat pathology, should be viewed with suspicion; and if this is coupled with a history of tinnitus and a family history of deafness—should be viewed with more than suspicion and should be considered as otosclerosis."

Until the last decade we had very little to offer the patient once a diagnosis of otosclerosis was made. The patient was subjected to numerous treatments such as Eustachian tube catheterization and politzerization; ear drum massages; nasal treatments both medical and surgical; dietary regimes of all sorts; endocrine regimes — orally, parenterally and by direct injection into middle ear cavity; Vitamin therapy and numerous other non-specific measures. The results as a rule were the same—the patient continued to have his hearing impairment and often was worse.

Yet, one must not be too dogmatic, nor should one condemn these measures. As recently as May, 1949, Lobel published his report on the treatment of 300 cases of deafness both of conductive and perceptive origin with a special Vitamin A preparation of high potency—50,000 units being injected intramuscularly or subcutaneously. He obtained "an appreciable gain in hearing function" in many patients. There have been no corroboratory reports to date and it would appear to be too early to pass any opinion on his report.

Surgical experimentation to create a new entry for sound waves to replace the obstruction at the oval window has been carried on for many years. As early as 1876 Kessel extracted an ankylosed stapes but the improvement was only temporary. Passow, Barany, Jenkins, Fraser and more recently Holmgren and Sourdille have attempted fistulization of the Labyrinth with no great success. The promintory and all the canals were fistulized in an attempt to create a new window and too frequently labyrinthine infection was a complication.

It was not until Lempert in 1938 published his "Improvement of Hearing in cases of Otosclerosis: A new one stage surgical technique" that a definite and positive advance was made. He introduced new instruments and new equipment. He emphasized the value of the direct approach to the middle ear by the Endaural route; and he also stressed the importance of good light and the extra special value of proper magnification. In 1940 he was able to report 120 fenestrations in which he reported 69 as "socially and economically rehabilitated."

The aim of this operation which is now referred to as the Fenestration operation is the creation and the maintenance of a new fenestra ovalis to replace the oval window whose function for the transmission of air borne sound to the cochlea was impaired.

The approach on this continent is via the Endaural route; in Europe the post auricular is still the most frequently used. The mastoid cells are only partially removed and the lateral semicircular canal is skeletenized. Very adequate exposure of the area is thus obtained and a small cavity for epithelialization is the result.

The fenestra (1 mm. by 3 mm.) is made on the dome of the lateral semi-circular canal and this is covered with a thin tympano—meatal-flap. Different operators have developed their own refinements of technique. Shambaugh uses a binocular microscope and emphasizes the value of continuous irrigation to clear the bone dust from the operative field and more recently he has developed a special type of headrest to be used post operatively.

House, in Los Angeles, uses a magnifying loop; a dry technique and utilizes a double blue line technique to create his fenestra. Recently Lampert reported a new technique in the formation of the fenestra whereby he thins the bony capsule and then uncaps it by incising it with a knife and lifting the bony cap, thus eliminating any possibility of bone dust entering the labyrinth; he then polishes the edges with a lead bur which he feels prevents osteogenesis.

The major problem, however, still remains and that is the maintenance of the fenestra nov-ovalis. A certain percentage, despite all precautions, continue to close over.

At this point I wish to outline and emphasize certain factors which must be taken into consideration when discussing the disability with the patient: because the patient seeking advice regarding this operation is vitally interested in these factors—namely, what are the results he can expect and what, if any, are the complications.

How then, do we decide what cases are the ones suitable for surgery? Generally speaking any individual from 15 to 60 with a progressive bilateral conduction type of deafness with intact drums, patent Eustachian tubes, with no serviceable hearing, in good health, with no ear, nose and throat infection and good nerve function is a candidate.

The nerve function is most important as our results bear a direct relation to its function and our choice of case is often guided by it. For practical purposes House divides his cases into Ideal, Borderline and Non-suitable cases.

The Ideal Case

Audiometrically the nerve loss must not exceed 10 decibels in the 512, 1024 and 2048 frequencies. A simple practical test can be made with a tuning fork (1024 tuning fork of magnesium alloy). The bone conduction is greater than the air conduction by at least 10 seconds. In such cases the results as a rule are good.

The Borderline Case

Audiometrically the nerve loss must not exceed 20 decibels in 512, 1024 D.V. frequencies and not to exceed 30 decibels in 2048 D.V. frequency. With a 1024 tuning fork B.C. is greater than A.C. by at least 5-10 seconds.

The Non-Suitable Case

Audiometrically the nerve loss is below 30 decibels in all frequencies or if with a tuning fork A.C. is greater than B.C. or A.C. equals B.C. or B.C. is less than 5 seconds greater than A.C. Occasionally one would or could operate in this type such as in the case of a young person with a "malignant" type of otosclerosis—where hearing loss is progressing at a rapid rate. The operation is done with the hope of preventing further nerve deterioration. The cause of nerve degeneration is not known; but in many cases the fenestration seems to inhibit its further degeneration. The cause of this is not known.

The above standards are not fool proof, but basically they are useful. Not every one possesses an audiometer; but careful tuning fork tests are almost as accurate. Anyone who can hear with a hearing aid and has conduction deafness of the type described may be a suitable case; especially if with a 1024 D.V. tuning fork his B.C. is greater than his A.C.

The amount of A.C. loss is not important if the cochlear nerve function is good. Yet there are authorities who will dispute this statement. If one ear has serviceable hearing as a rule one does not advise an operation in the non-serviceable ear unless the nerve loss is rapid or unless the tinnitus is of the almost unbearable type. In making the decision to operate in the average bilateral involvement the poorer of the two ears is as a rule selected. Complications do occur and in discussing the operation with the patient it is wise to advise him about these possibilities:

1. Temporary facial paralysis occurs in about 1% of cases.

2. Post-operative vertigo may be fairly severe for 2-5 days, but as a rule it subsides in 4-6 weeks; but some may be troubled with it for a long time.

3. Otitis media with or without a perforation may occasionally complicate the picture and impair the result.

4. Serious labyrinthis is a very rare complication.

5. A persistent mucoid discharge may prove to be troublesome in about 20% of the cases. This is not as persistent a complication since the decrease in the size of the mastoid cavity.

6. Bony or fibrous closure if it does occur will do so within the first six months. However, if the serviceable hearing is maintained for six months the likelihood is that it will remain patent (90% of cases). The case of closure can be revised after one year if there was a serviceable hearing improvement in the first six weeks, that is, if the nerve function is still good.

The results of the fenestration operations are good in properly selected cases. In the ideal case we can expect a restoration of the hearing to a serviceable level and the patient can carry on all ordinary business and social pursuits without the use of an aid. The reports in the literature suggest that 75%-90% of "ideal" groups can expect restoration of serviceable hearing. In the borderline cases the odds lessen and the percentages run from 30%-55%.

In a small percentage of cases the result may not last due to the closure of the fistula as a result of new bone growth from the periosteal and endosteal layers about the margins of the fistula. There has been no positive method devised as yet which will prevent this new growth.

If it does close one can operate again but one should wait about one year; that is, providing the ear had shown a serviceable return of hearing after the operation; or one can operate on the other ear, that is, providing a hearing aid works in the previously operated ear. Recently Max Pohlman has reported restoration of hearing utilizing a synthetic drum membrane and a nylon bristle acting as ossicles. I know of one such case that had both ears fenestrated with a poor result and is carrying on with a Pohlman insert. One would wonder if these were cases of true otosclerosis.

A very important favorable result that may occur even though the hearing acuity does not come up, is the tinnitus may decrease or even disappear in a large percentage of the cases.

One may ask "why not recommend a hearing aid?" The aid does bring the hearing up to a serviceable level in otosclerosis and therefore why subject the patient to a major operation which cannot guarantee a 100% result and which operation hospitalizes him, keeps him from work and is a definite drain on his pocketbook. For the answer to this I wish to quote Julius Lampert:

First: The operation if successful restores physiological hearing function, whereas the aid amplifies sound without improving the function.

Secondly. The progression of the hearing loss is retarded, whereas with aid it continues.

Thirdly: Tinnitus is eliminated in 80% of the cases following a fenestration, whereas there is no change in an aid.

Fourthly: Voice sounds are normal and not distorted as with an aid.

Fifth: Group conversation is restored in the successful case but is difficult with an aid.

Sixth: And not least important is the change in the mental outlook of the patient.

Summary

(1) Clinical otosclerosis is a progressive bilateral conduction type of deafness which in the later stages is also associated with a perception loss.

(2) Diagnosis is based on the history; the clinical findings of a normal drum and patent Eustachian tubes and the audiometric and tuning forks findings of a conduction deafness in the early stages.

(3) Medical treatment has to date been found wanting.

(4) The fenestration operation offers the first positive and successful approach to the problem of otosclerosis.

(5) Serviceable hearing can be expected in approximately 80% of cases in the ideal group.

(6) Tinnitus is relieved in approximately 80% of the cases.

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ORTHOPEDICS

Injuries of the Semi-Lunar Cartilages

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Semi-lunar cartilage injuries form one of the major causes of Internal Derangements of the knee joint.

The term Internal derangement of the knee joint was first coined in 1784 by William Hey, an English surgeon who practised in Leeds. Hey used the term to denote any alteration of the knee joint which prevents the condyles of the femur from moving truly in the hollows formed by the semi-lunar cartilages and depressions of the tibia. The modern term is used to include intra-articular lesions due to trauma, occurring in a previously healthy joint. It is also customary to include injuries of the collateral ligaments in this group.

Next in frequency to collateral ligament injuries are those of the semi-lunar cartilages. Semi-lunar cartilage injuries occur more frequently on the left side than on the right. They are rare in children, but are common between the ages of 18 and 30. They are much more frequent in males and occur frequently in competitive sports.

The injuries of the semi-lunar cartilages are sustained on the flexed knee. When the flexed knee is exposed to a rotation strain, one or other of the semi lunar cartilages may be injured, due to the grinding action of the femur on the tibia. The position at which the strain is applied to the cartilage depends on the angle of flexion of the joint at the moment of injury. The greater the flexion at the time of injury, the more posterior will be the subsequent lesion. This is the basis of McMurray's test. According to McMurray, at least 75 per cent of cartilage injuries occur in the posterior half of the joint.

The actual damage which the cartilage may suffer varies much. There may be:

(1) A longitudinal split, separating the posterior end of inner half of cartilage.

(2) The whole cartilage may be split longitudinally into two—the so-called Bucket handle deformity.

(3) A longitudinal split separating anterior end of inner half of cartilage.

(4) Either posterior or anterior horn may be torn from its tibial attachment.

(5) Portions of the cartilage may become completely detached—form loose bodies.

(6) Marginal detachment of the cartilage may occur.

The cartilage, if partially displaced, may occasionally protrude outwards, but usually projects internally into the interconylar notch.

The Medial Semi-lunar Cartilage—In about 80 per cent of cases of semi-lunar cartilage injury it is the medial one which has been damaged.

A typical history is that of a football player who makes a sudden turn while running and as his weight is thrown on the abducted and flexed knee, the joint suddenly "gives way" and he falls to the ground with severe pain over the inner side of the knee. On attempting to get up he is unable to completely extend the knee. Extension is found to be limited by 10 degrees to 30 degrees. There is an elastic block just short of full extension. This is what is meant by "locking in extension." Within

a few hours swelling develops as a result of bleeding and effusion in the joint—due to the associated traumatic synovitis accompanying the cartilage injury.

The "locking in extension" is the most characteristic sign, and in an acutely injured knee is almost pathognomonic of dislocation of a semi-lunar cartilage. It may not be present if the cartilage is merely torn but not dislocated. In such cases, however, tenderness and pain are localized over the semi-lunar cartilage along the joint line. The tenderness may be greater over the anterior or posterior portion of the joint, but is always present over the middle of the tibial collateral ligament—that is, at the attachment of the deep fibres of the tibial collateral ligament to the medial meniscus.

X-rays of the joint are negative as the cartilage does not show in the film.

When the diagnosis is in doubt, it may sometimes be confirmed by air arthrography, i.e., an X-ray picture which is taken after air has been injected into the knee joint. By this procedure the tear in the cartilage may be visualized.

Of far more value than air arthrography is McMurray's test. T. P. McMurray who recently passed away (Nov. 16, 1949), will probably best be remembered in surgery for this test. In performing the test, the patient lies supine on the examining table. The surgeon stands on the side of the injured limb. The foot is grasped firmly and the knee flexed completely, so that the heel approaches or touches the buttock. The foot is now rotated externally and the leg abducted at the knee, whilst the joint is slowly extended. With the alteration in the angle of the joint, any loose portion of the medial semi lunar cartilage is caught between femur and tibia, and the sliding of the femur over the injured portion of the cartilage is accompanied by a definite click and pain which the patient states is similar to the feeling experienced when the knee gave way. In this test it is to be noted that the abnormal click is painful.

Treatment of Acute Injury of the Medial Semi-Lunar Cartilage

The treatment is conservative. If swelling is marked, the knee is aspirated. If the knee is locked, an effort may be made to reduce the dislocation. The method of manipulation used is to forcibly flex the knee, while the surgeon's forearm is in the popliteal fossa. This tends to push the upper end of the tibia forward and may dislodge the cartilage. The leg is rotated medially and laterally and slowly extended. One may hear a "click" as it is reduced. No anaesthetic is used.

If the first attempt at manipulative reduction is not successful, a second or third attempt should not be made—so as to avoid further damage to the knee. At this stage, some surgeons operate at once to remove the injured cartilage. However, if the patient is put to bed with a firm compression bandage to the locked knee, in many instances the locking will disappear in a day or so.

Test of reduction—Patient is able, by his own active efforts, fully and painlessly, to extend the knee and maintain it extended.

After manipulative reduction, or if no reduction is necessary, patient is kept in bed for two weeks. During this period, a firm compression bandage over a thick layer of cotton wool to the knee is maintained for the first week to prevent or diminish effusion. Then movement, active and passive, and also massage, is employed for a further week. Then patient is allowed out of bed and starts to walk. No games allowed for two months—this is to avoid abduction or lateral rotation strain. During this period, inner border of shoe is elevated ¼ inch.

After such treatment there may be no further trouble. In some cases, however, recurrent dislocation of the cartilage occurs.

Recurrent Dislocation of the Medial Semi-Lunar Cartilage

Any semi-lunar cartilage which has been out more than once is a **recurrent** one. In recurrent cases, the symptoms are not so severe, and the locking is often transient in nature; or the patient may have learned to reduce the semi-lunar cartilage himself. The severity of the symptoms tend to decrease with the frequency of the dislocation. Some of the chronic cases in which the cartilage slips out very frequently develop **chronic synovitis** and a variable amount of osteo arthritis. In these cases the history is the most important factor in diagnosis.

If the patient is seen in the interval between attacks, there may be very few physical signs on which the surgeon can base his diagnosis and it must depend largely on the patient's history. A careful history is therefore important, noting details of onset of symptoms, nature of injury if any, and character of symptoms. The patient's history of knee "giving way" and of "locking" should be carefully queried. In referring to locking the patient not uncommonly means stiffness rather than "locking."

Treatment of Recurrent Dislocation

(1) Conservative treatment is advised only if there is some contra-indication to operation. It consists in wearing an elastic knee-cap or elastic bandage for a few weeks until synovitis subsides. The patient is instructed to walk with "toes in." A knee cage which is sometimes recommended is of little value.

(2) Before operation is decided upon, manipulation under anaesthesia should be performed. Intra-

articular adhesions often form as result of damage to the semi-lunar cartilage or infra patellar fat pad and considerable benefit may result from breaking down such adhesions. Indeed manipulation may cure the condition.

(3) The treatment in general is **operative removal** of the torn or displaced cartilage. Quadriceps exercises before and after operation is an important adjunct to prevent a weak knee. After the cartilage has been completely removed, if the knee is normal in other respects, one may expect full function in 6-12 weeks. If the semi-lunar cartilage is not removed it will lead to damage of the hyaline cartilage with resultant osteo arthritis. If the affected cartilage is incompletely removed, there is recurrence of symptoms. Good results are obtained in about 75% of cases of cartilage removal. Some of the imperfect results of operative treatment may be traced to (1) relaxed collateral ligaments; (2) torn cruciate ligaments; (3) osteo arthritis.

In such cases, however, the operation relieves the tendency of the knee to lock and also removes a causative factor in arthritis.

Injury to the Lateral Semi-Lunar Cartilage

The lateral semi-lunar cartilage is subject to the same types of injury as the medial, but it is injured only about 1/5 as often. The lesions in the cartilage resemble those in the medial cartilage, but the symptoms are less clear cut. Locking, which is common with lesions of the medial cartilage, is rare with those of the lateral. Pain and tenderness are not so consistent as in lesions of the medial cartilage and subsequent displacements are not as a rule followed by synovitis of the joint.

The typical history is that the knee gives way suddenly with a feeling of weakness or slipping on the "outer side," followed by a slight tenderness on pressure over the anterior horn of the cartilage or over the fibular collateral ligament along the joint line. Accompanying this, there is often a loud **click** which can be felt and heard and which occurs most typically in the last few degrees of **active** full extension of the joint but does not occur when the movement is carried out passively. Additional evidence can usually be elicited by McMurray's test as performed for the medial cartilage with the difference that on examination the foot is rotated **inwards** and the leg adducted at the knee.

Treatment: Applies as for medial semi-lunar cartilage injuries.

Cysts of Semi-Lunar Cartilages

Occasionally, after an injury to the semi-lunar cartilage, a multi locular cyst develops in the cartilage and the cartilage increases in size until it interferes with the movement of the joint and causes pain. These cysts are more frequent in the lateral semi-lunar cartilage. They are the only form of cyst which occurs actually in the knee joint line. Treatment is excision of the cartilage with the cyst.

MEDICINE

Vitamin B_{12} — A Review

R. T. Ross, M.D.*

The therapeutic effect of liver in pernicious anaemia has been known since the original description by Minot and Murphy[1] in 1926. Since that time there has been continuous investigation in an attempt to isolate the active principle. The available commercial liver extracts are crude concentrates containing the active principle and are standardized in units by clinical trial, one U.S.P. unit having been defined as that amount of liver extract required daily to produce a satisfactory clinical and haematological response in a case of Addison's pernicious anaemia. Clinical trials with supposedly pure fractions have been infrequent and the results equivocal, while no method of bioassay has been described that has helped the work of fractionation.

Recently Rickes et al[2] have shown that further purification of commercial liver extract was possible. They isolated a substance which West[3] demonstrated to be clinically effective. This fraction was examined further by Shorb and Briggs and found to be microbiologically active for the growth of Lactobacillus Lactis Dorner. This growth factor was named L.L.D. factor and appeared to be related to the activity of commercial liver extracts.

Further purification (by Rickes et al[2]) of clinically active liver fractions led to the isolation of a crystalline compound in minute amounts that was highly active for the growth of Lactobacillus Lactis. This was called Vitamin B_{12}. The reasons of the original investigators[2] for so naming the substance are:

"The long expression 'anti-pernicious anemia principle' is not advisable, largely because the biological role of this compound in the treatment of pernicious anaemia or other diseases is yet to be learned. The term 'L.L.D. factor' implies a biological limitation and was used originally to name an activity. The biological activities of crude materials are frequently found later to be due to several chemically related entities, as exemplified

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in the vitamin and antibiotic fields. A trivial name based upon source or activity is undesirable. The name Vitamin B_{12} has not been used in the B series and connotes only nutritional significance. We may in the future wish to designate a name based upon chemical structure[2]."

To establish some standard of potency, the originally selected liver concentrate was given an arbitrary designation of 1000 L.L.D. units per milligram. The pure crystaline B_{12} was found to have a potency of 11,000,000 L.L.D. units per milligram. Further examination of a number of commercially available liver extracts has revealed that the content of Vitamin B_{12} varies from 1.2 to 14.0 micrograms per cubic centimetre. Each of these samples bore a label claim to contain 15 U.S.P. injectable units per cubic centimetre.

Working on the premise that the active ingredient of liver extract was Vitamin B_{12}, many investigators have attempted to determine a quantitative relationship between a unit of liver and a microgram of B_{12}. At this time it appears that one U.S.P. unit of liver is probably equivalent to one microgram (1/1000 of a milligram) of Vitamin B_{12} in both microbiological and clinical potency.

In an attempt to determine the optimum dose of B_{12}, West[3] has given the drug to a small group of patients with pernicious anaemia. To four patients he gave a single intramuscular dose equivalent to 2 to 4 micrograms. These four all showed maximal haematological responses. To three cases receiving the equivalent of 1 microgram of B_{12} the haematological response was less than optimal. These results which have subsequently been corroborated indicate that the U.S.P. unit of refined liver is equivalent to something between 1 and 3 micrograms of Vitamin B_{12}.

The minute amount of this compound required for microbiological growth and clinical response places Vitamin B_{12} among the most potent microbiologically active compounds.

Nature and Source

From the information available at the present time it is known that Vitamin B_{12} is a red, crystalline compound with a molecular weight of about 1600. It contains phosphorous, nitrogen and cobalt, and is ineffective biologically and clinically apart from the cobalt. It is soluble in water and alcohol and insoluble in ether and chloroform. Identified sources of the substance are milk powder, beef and hog liver, extract of beef muscle. It is also present in the culture broth of several micro-organisms, among them streptomyces griseus, the organism from which commercial streptomycin is obtained.

Also Bethel[4] has demonstrated large daily fecal excretions of Vitamin B_{12} from 4 cases of untreated pernicious anaemia.

It is interesting to note that at the same time as the publication of Rickes[2], Smith[5] by a process of chromatography identified a pure substance with properties very much like those of Vitamin B_{12}. The base substance used was ox liver and the purified fraction derived from it appears to be clinically effective in doses of about 2.0 micrograms per day in pernicious anaemia.

Clinical Results With Vitamin B_{12}

In addition to the published results of West[3], Hall and Campbell[6, 7] have reported on the effect of parenterally administered B_{12} to 11 cases of pernicious anaemia. Having varied the dose considerably, they conclude that approximately 1.0 microgram of B_{12} is equivalent to 1.0 U.S.P. unit. They were not able to demonstrate any significant difference in the response to 1.0 microgram per day from the response to 5.0 micrograms every 5 days, or 25 micrograms once a week.

Clinically they noted improvement in strength, mental alertness and appetite, a gain in weight and disappearance of glossitis. Three patients with peripheral neuritis but without involvement of the spinal cord showed improvement or disappearance of paresthesias in from one to one and a half months. Five of six patients with peripheral neuritis and combined degeneration of the cord improved; in 3 the improvement was very rapid. They also observed serial bone marrow changes with complete reversion from megaloblastic to normoblastic within 72 hours.

Bortz[8] has reported the effect of Vitamin B_{12} on five cases of pernicious anaemia. He has varied the dose from 10 to 25 micrograms per day. These doses have contributed nothing towards establishing the optimum daily doses. However, in all his cases the responses were maximum and the patients all experienced a feeling of well being, increased vitality and strength very shortly after starting treatment. Red blood cell regeneration in all patients was maximum and the improvement in central and peripheral nervous system signs and symptoms was as good as that expected had the treatment been liver extract.

Also, Schieve et al[9] report the cases of two patients being treated with folic acid for pernicious anaemia, who developed intensely sore tongues. Within 5 days after a single intramuscular injection of Vitamin B_{12}, all signs and symptoms of lingual pathology disappeared.

They report further that in 7 new cases of pernicious anaemia treated with B_{12}, the lingual manifestations improve very rapidly, no patient having symptoms after the fifth day of treatment.

Berk et al[10] have reported the case of a patient with pernicious anaemia who was being treated with folic acid due to a sensitivity to injected liver extract. While on folic acid therapy, the patient experienced an haematological and neurological relapse but responded completely to daily injec-

tions of Vitamin B_{12}. There was no further evidence of sensitivity reactions with the B_{12}.

On the other hand, Day, Hall and Pease[11] report an excellent haematological and clinical response from the parenteral administration of folic acid in a case of megaloblastic anaemia of pregnancy in which the previous administration of liver extract and of Vitamin B_{12} had failed to elicit any response.

The most comprehensive study to determine the minimal daily effective dose of B_{12} has been done by Jones, Darby and Trotter[12]. They have treated 11 cases of pernicious anaemia and found that less than 0.75 micrograms daily in doses at intervals of several days did not establish or maintain blood values as high as expectancy curves would indicate were maximum. Parenteral daily doses of 1.0 microgram or greater promoted maximal erythropoiesis in all cases. They also found that the optimum initial daily dose is approximately 3.0 micrograms.

Further support is added to the premise that 1.0 microgram of Vitamin B_{12} is of equal potency to 1 U.S. P. unit of liver extract when one recalls that maximum reticulocytosis and erythropoiesis is obtained from an initial daily intramuscular dose of liver extract of 3.7 U.S.P. units as determined by Jacobson and SubbaRow[13].

Murphy[14] determined the optimal initial daily dose of liver to be 2.1 units.

Means of Administration

Up to the present time the only successful manner of treating pernicious anaemia with Vitamin B_{12} has been to administer the drug by intramuscular injection. However, a few reports are available on the results of giving the drug by mouth.

Spies[15] has found that in some cases of pernicious anaemia, when B_{12} is given by mouth in 500 to 1500 microgram doses daily, there is an haematological response. The unpredictability of response, the huge doses required, and the expense of this form of treatment make this procedure completely impractical at this time.

Hall, Morgan and Campbell[16] have also given the drug by mouth but have given normal human gastric juice at the same time. They have found that the oral administration of 25 to 35 micrograms of Vitamin B_{12} alone per week is ineffective in pernicious anaemia. Also, as has been known for some time, they found that 150 ccs. of normal human gastric juice is by itself devoid of haematopoietic activity when given to patients with pernicious anaemia. However, when normal human gastric juice is administered simultaneously with or within two hours of oral administration of 5 micrograms or more of Vitamin B_{12}, an haematopoietic response occurs. They also determined that at least 25 ccs. of gastric juice must be given with each 5 micrograms of B_{12} to obtain an optimum haematopoietic response.

Conclusions and Summary

1. Crystalline Vitamin B_{12} has been isolated from commercial liver extracts and is probably the substance responsible for the clinical efficacy of the latter.

2. Vitamin B_{12} in micrograms appears to bear an almost linear relationship in potency to liver extract in U.S.P. units.

3. To date there is no reported case of Addisonian pernicious anaema in which Vitamin B_{12} has been ineffective therapeutically or has caused a sensitivity reaction. It has been successfully administered to a patient already sensitive to a number of commercial liver extracts.

4. The haematopoietic activity of oral Vitamin B_{12} is enhanced by the simultaneous administration of normal human gastric juice, but is not as great as when the vitamin is given parenterally. It is suggested, therefore, that the extrinsic factor may be identical with, or closely related to, the antipernicious anaemia principle of liver, which is itself presumably identical with Vitamin B_{12}.

It is also suggested that the intrinsic factor is necessary for optimal utilization or absorption of the extrinsic factor B_{12} from the gastrointestinal tract but probably does not combine with it to form a third and different substance.

5. The initial daily dose of Vitamin B_{12} to produce an optimal haematological response is about 3.0 micrograms. The daily maintenance requirement administered at regular intervals appears to be 1.0 microgram.

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CASE HISTORIES—SURGICAL

Diffuse Toxic Goitre, Grave's Disease Subtotal Thyroidectomy

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This is the fourth of a series of Case Histories which will appear in the Review each month. The purpose of these publications is not to present rare or unusual cases but rather to consider the routine management of common surgical conditions.

Case No. A-5478, Mrs. S. M., St. Boniface Hospital. Color, white. Age, 36 years. Occupation, housewife. Date of admission, May 21, 1948. Date of operation, May 31, 1948. Date of discharge, June 5, 1948.

Entrance Complaints

1. Headaches, 4 years. 2. Cannot sleep at nights, 3 years. 3. Very nervous, 2 years. 4. Loss of weight, 2 years. 5. Feels heart pounding, 2 months. 6. Hands tremble, 1 month.

Present Illness

Early in 1944 the patient began experiencing intermittent headaches which would last from 12 to 24 hours each and occur once or twice a week. The headache was actually a sense of pressure over the top of the head and was never associated with nausea or vomiting. Aspirin would not relieve the headache. The discomfort in her head would very frequently keep her awake during the night, and it was to this loss of sleep that she attributed the nervousness, irritability and easy fatiguability which she began to experience early in 1946.

Beginning about June, 1947, she frequently experienced a choking sensation in her neck when in bed at night, which was only relieved when she got up and walked around. Her weakness and tiredness gradually increased and by December, 1947, she could not walk up stairs without sitting down several times to "catch her breath" and give her legs a rest. In February, 1948, the patient had all her teeth pulled. This seemed to aggravate her irritability so that she could not stand any noise from the children or even people talking in the house.

In March, 1948, she began to feel her heart "pounding like a hammer" in her chest. In April, 1948, she noticed that her hands were very shaky and perspired freely.

Since early 1946 the patient has decreased in weight from 140 pounds to 108 pounds.

Inventory By Systems

Eyes—Vision good, no diplopia or blurring of vision.

Ears—Hears well, no vertigo or tinnitus.

Respiratory—Infrequent "head colds" and sore throats. No chest pain, cough, expectoration or hemoptysis. Dyspnoea on slight exertion, such as climbing stairs, since about December, 1947.

Cardio-vascular—No history of rheumatic fever or syphilis. Palpitation of heart, even at rest, since March, 1948. No precordial pain or dependent oedema. Dyspnoea, as above, since December, 1947.

Gastro-intestinal—Appetite only fair since 1946. No dyspepsia, nausea or vomiting. No abdominal pain. Bowels regular. No melena. No history of jaundice.

Genito-urinary—Frequency (about every 3 hours). Nocturia (1-2x) when she drinks before going to bed. No burning or pain on micturition. No blood in urine.

Menstrual—Menarche at 14 years of age. periods regular, every 28 days. Duration of flow, 3 to 4 days. Amount of flow, scant. No dysmenorrhea or dyspareunia. No intermenstrual bleeding or discharge.

Obstetrical—Para ii; Gravide ii. First pregnancy (1939)—pregnancy and labor uneventful. Second pregnancy (1943)—pregnancy and labor uneventful. Both children alive and well.

Nervous System — Headaches, tremors and weakness as in history of present illness.

Metabolic — definite heat intolerance since February, 1948. Does not like a hot, sunny day, and feels very uncomfortable cooking on a hot stove. Loss of weight (140 pounds to 108 pounds) since 1946.

Past History

Measles and mumps during childhood.
No significant previous illnesses.
No surgical operations.
No accidents.

Family History

Mother—alive and well, 65 years of age.
Father—alive and well, 70 years of age.
Two brothers—alive and well, ages 39 years and 25 years.
One sister—always sick (cause unknown), age 37 years.
No family history of tuberculosis, cancer, diabetes, heart disease, epilepsy, mental disease, etc.

Physical Examination

General impression—The patient is a very thin, haggard-looking female, who appears very tense and jittery and moves around continually in bed.
Head and Neck:
Cranial nerves—Intact.

Eyes—Lids, conjunctivae, corneae and lenses normal. No widening of palpebral fissure. Very slight lid-lag evident. No exophthalmos or weakness of convergence. Pupils equal and react well to light and accommodation.

Ears—External ears, canals and drums normal.

Nose—No obstruction; mucosa clear.

Lips, Tongue, Gums—Normal.

Teeth—Edentulous.

Throat—Tonsils small. Pharynx clear.

Larynx—Both vocal cords move freely.

Neck—No cervical lymphadenopathy. No venous engorgement. Thyroid gland firm, mobile, and shows moderate symmetrical, smooth enlargement of right and left lobes. Slight soft bruit audible over gland.

Chest:

Mammae—Small, flat. Nipples erect; no discharge. No lumps visible or palpable.

Heart—Apex in 5th interspace, 3½ inches from midline. Heart regular, sounds loud and clear. Rate 110 beats per minute. Soft systolic murmur audible over pulmonary area. Blood pressure 150/80.

Lungs—No deformity of thoracic cage. Movements symmetrical and equal. Tactile fremitus good throughout. No area of dullness. Breath sounds clear, no adventitious sounds.

Abdomen—Scaphoid in appearance, skin loose and wrinkled. No areas of tenderness, guarding or rigidity. Liver and spleen not palpable. No masses palpable. No herniae. Reflexes present and equal.

Pelvic Examination — Introitus lax. Uterus small, shape and position normal. No masses or tenderness in fornices. Cervix appears healthy.

Rectal Examination—No abnormal findings.

Extremities:

Upper—No deformities. Movements good. No clubbing of fingers. Palms warm and moist; skin smooth. Fine tremor of fingers with arms outstretched.

Reflexes:	Right	Left
Biceps	++	++
Triceps	++	++
Supination	+	+

Lower—No deformity or limitation of movements. No varicosities or dependent oedema. Some weakness of Quadriceps muscles.

Reflexes:	Right	Left
Knee	+++	+++
Ankle	++	++
Plantar	V	V

Clinical Laboratory Findings

Urinalysis—Urine turbid, amber colored, acid in reaction. Specific gravity 1.022. Albumin, 0. Sugar, 0. Microscopic, 2-3 red cells per h.p.f. 4-5 pus cells per h.p.f. No casts.

Blood—Red blood cells, 4,240,000. Hemoglobin, 85%. Color Index, 1.0. White blood cells, 9,350. Polymorphonuclear Neutrophils, 74%; Lymphocytes, 26%.

Blood, Wasserman—Negative.

Hippuric Acid test of liver function, 100%.

Basal metabolic rate, +32.

Chest X-ray—Bony thorax is normal. Diaphragms are normal. Heart and great vessel shadows are normal. Lung fields are clear.

Pre-operative Diagnosis

Diffuse toxic goiter—Grave's Disease.

Indications for Operation

This patient manifested unmistakable clinical symptoms and signs of thyrotoxicosis, and at the present time subtotal thyroidectomy after suitable preparation is the treatment of choice for this condition.

Pre-operative Care

The patient was admitted to hospital where she lay resting in bed for ten days before her operation, and received the following medications:

1. High caloric diet.
2. Frequent glucose drinks.
3. Multicebrin (vitamins) i b.i.d.
4. Lugol's solution min. xii t.i.d.
5. Phenobarbitol gr. ½ q.i.d.

During her pre-operative preparation her weight increased 2 pounds, she felt much improved symptomatically, her pulse rate decreased from 110 to 72 per minute and her blood pressure from 150/80 to 130/80.

Detailed Description of Operative Technique and Operative Findings

Position—Supine. Sandbag is placed behind the shoulders so as to render the goiter prominent.

A frame with screen adjusted to isolate the head and face. Skin painted with merthiolate. Draped. Local Anaesthetic. About 50 cc. of 1% novocaine solution (without adrenalin) was first injected subcutaneously along the line of the proposed incision, then in radiating lines towards the upper poles and finally injected deeply in the region of the upper poles.

Incision—A piece of catgut is forcibly indented in the skin of the neck to outline the incision. In this case the goiter being somewhat prominent, the incision was placed about an inch and a quarter above the Xiphi-sternal notch. A transverse collar incision extending from the outer border of one sterno-mastoid muscle to a corresponding point on the opposite side was made. The skin and platysma were completely divided. The upper flap consisting of the skin, superficial fascia, and platysma was dissected upwards by means of gauze dissection and snipping here and there of fibrous bands with scissors up to the notch in the thyroid cartilage. The lower skin edge was dissected in a similar manner in a downward direction for about half an inch. All small bleeders were ligated with

000 chromic catgut. The two skin flaps were now retracted by means of hook retractors. The deep cervical fascia covering the pre-tracheal muscles was picked up with 2 hemostats, one on each side of the midline, and divided in a vertical direction to the entire length of the exposure. The anterior jugular veins were not cut. The right strap muscles were retracted laterally to visualize the right lobe of the gland. The index finger was passed under the strap muscles on the right side, and the right lobe was exposed in its capsule and gently mobilized. A thyroid seizing forceps was hooked into the body of the right lobe and by gentle traction almost the entire lobe was dislocated from its bed and lifted into the wound. The middle thyroid vein was grasped between 2 hemostats, divided, and ligated. A pair of blunt curved artery forceps were now insinuated gently into the space between the upper pole and the trachea and the blades opened to free the upper pole. Three heavy forceps were applied to the upper pole of the gland so as to include the superior thyroid vessels. The pole was divided between the middle and the lower forceps. A chromic catgut No. 1 ligature was applied above the uppermost forceps and tied firmly as the latter was removed. A second ligature was then applied and the lower forceps was removed. The right lobe was then pulled over to the left, and a number of Halstead hemostats were placed in pairs along the most posterior part of the capsule of the gland in a line towards the inferior pole. The blood vessels were divided between the forceps and individually ligated with 000 chromic catgut. The lower pole was now elevated and the inferior thyroid veins were clamped, cut and ligated. The inferior thyroid artery was not isolated separately. Several artery forceps were now thrust into the substance of the gland and the thyroid divided in front of them. The suspensory ligament of the thyroid was next dissected out from the front and sides of the trachea, clamped, divided and ligated. A long curved artery forceps was insinuated under the posterior aspect of the isthmus from its upper border until it was seen to emerge at the lower border. The blades of the forceps were separated, the isthmus was then clamped between two large hemostats; the right hemostat was gradually rotated to the right so that the isthmus could be separated gently from the front of the trachea. Forceps were now thrust into the gland from the left side parallel to the trachea, cutting away the thyroid with gentle strokes of the scalpel in such a way as to leave a portion of thyroid tissue in the region of the inferior thyroid artery, the recurrent laryngeal nerve and the parathyroids for protection against injury. About one-seventh of the gland was left in its bed. The blood vessels were now ligated with chromic catgut 000. The edges of the capsule were then sewn over by means of a continuous interlocking 000 catgut suture. The wound was now inspected for any oozing. A small hot saline pack was inserted into the cavity left. The left lobe of the gland was then mobilized and resected in a similar manner. The hot pack was now removed, the wound irrigated with saline, the sternothyroid muscles approximated to cover the trachea by a few interrupted chromic catgut 000 sutures. The sternohyoids with the fascia covering them were then approximated by similar interrupted fine sutures of catgut 000. The neck was then flexed and several interrupted chromic catgut 000 sutures were used to approximate the cut edges of the platysma. The skin was closed by a fine continuous over and over silk suture. No drain was used. A thyroid dressing was applied. At the end of the operation, the anaesthetist visualized both vocal cords and reported free movement.

Anaesthetic

Pre-medication—Tuinal gr. iii the evening before operation. Morphine gr. 1/6 with atropine gr. 1/150 one hour pre-operatively.

Before induction—Temperature 97.3° F. Pulse, 72. Respiration, 21. Blood pressure, 130/80.

Induction—Satisfactory.

Maintenance—Easy.

Agents—Nitrous oxide cyclopropane oxygen.

Technique—Closed.

Haemorrhage—Minimal.

Stimulants—None.

Post-operative condition—Good.

Gross and Microscopic Description of Tissues Removed

Thyroid gland:

Gross—2 roughly oval, even-sized thyroid lobes of large hen's egg size, measuring 6 cm. long, by 4 cm. wide, by 3 cm. thick. Surface of lobes moderately lobulated. Combined weight, 95 gms. Cross section does not show the normal colloid appearance, but instead an opaque-looking, meaty tissue of light pinkish-yellow color. No adenomata present.

Microscopic—The acini are small, medium and large in size, the former two predominating. The colloid is dense and acidophilic, at times vacuolated. Much of the acinar epithelium is cuboidal and low cuboidal, and there are traces of hyperplasia with some tufting of the epithelium. Lymphocytic infiltration is scant and fibrosis minimal. Vascularity is increased and small haemorrhagic areas are seen here and there throughout the gland.

Final Diagnosis

Diffuse toxic goitre—Grave's Disease.

Progress Notes

May 31, 1948—Immediate post-operative orders:

1. Intravenous 1000 ccs. 5% glucose in saline, with one ampoule of sodium iodide daily for 2 days.

2. Intravenous 1000 ccs. 5% glucose in distilled water with one ampoule of sodium iodide daily for 2 days.

3. Morphine gr. ¼ for pain, when necessary.

4. Ice cap to neck.

Patient spent an uneventful post-operative day and night except for vomiting up 100 ccs. of clear fluid on one occasion.

June 1, 1948—Temperature 103° F. Pulse, 110. Respiration, 24. Patient looks well. Allowed up in chair. No hoarseness of voice. Started on penicillin 30,000 units intramuscularly every 4 hours. Lugol's solution min. x b.i.d., phenobarbitol gr. ½ q.i.d., and multicebrin b.i.d. resumed.

June 2, 1948—Temperature 102° F. Pulse, 90. Respiration, 20. Patient looks and feels well. Walking about for short intervals. No complaints.

June 3, 1948—Temperature 101° F. Pulse, 85. Respiration, 20. Patient feeling very well. Sutures removed. Wound looks good.

June 5, 1948—Temperature 98.4° F. Pulse, 70. Respiration, 20. Patient discharged. Advised to continue with Lugol's solution min. v daily and phenobarbitol gr. ½ for one month.

Condition on Discharge

Improved.

Follow-up Notes

June 10, 1948—Patient returned to the country. Has not been seen since the operation.

CASE HISTORIES—MEDICAL

Lederer's Anemia

Report of a Case and Brief Review of the Disease

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Case Report

This was a girl, born in 1938, who was admitted to St. Boniface Hospital, May 2nd, 1949. She had an uneventful delivery and early childhood. Her mother thought that perhaps she was always somewhat paler than her siblings. For some years she had had bouts of slight headache, nausea and vomiting that occurred about once a month, more often in summer than in winter, generally came on in the evening and lasted a few hours. She did not become jaundiced after any of these attacks, nor did she show any purpuric spots or free bleeding. Tonsillectomy was done in April, 1948, and apart from some minor postoperative bleeding, was uneventful. She did well at school and was in grade 5 at the age of 11. She played with the other children and appeared to be a normal child.

On May 1st, 1949, at school, she experienced a severe frontal headache, and some vague upper abdominal discomfort. She fainted at school and was sent home. Her mother thought she was quite pale when she arrived home. That evening she vomited 6-7 times, vomiting up clear, greenish fluid. In the early afternoon of May 2nd she was taken to see her physician. She fainted in his office, and was admitted directly to hospital.

On admission a very tired-looking girl was seen. She was extremely pale, her palpebral conjunctiva and her lips were waxy in hue; the palms of her hands were almost paper-white. Her temperature was 100° F. by mouth; respirations 24/min. and pulse rate 140/min. Examination of her thorax was negative. Her heart rate was 140/min. and regular. A harsh systolic murmur was heard over the whole precordium, loudest at the mitral area. Blood pressure was 90/70.

Abdominal examination disclosed the liver edge 3 cm. below the costal margin, moderately firm, and not tender. The spleen palpable 2 cm. below the costal margin was moderately firm and not tender. Many purpuric spots were seen on the limbs, neck and upper chest. No lymphadenopathy was found. Hemoglobin estimation done at once was 20%, and the red cells were one million. She was given 300 cc. of blood at once. Blood was Group 0, RH negative.

The clinical impression at this time was that this might be an acute, leucemia, or subacute bacterial endocarditis. A blood culture was taken, which subsequently was negative; and she was given penicillin. A marrow aspiration from a lumbar spinous process disclosed marrow that showed normo-blastic hyperplasia but was otherwise negative, and was considered strong evidence against this being a case of leucemia. The possibility of this being an hemolytic process, either acute, or an acute exacerbation of a chronic hemolytic anemia was considered, and further studies were carried out.

Blood Wassermann was subsequently reported as negative. X-ray of chest was negative. Complete blood count revealed the following: Hgb 20%; Red cells 1.1 million; White cells 8.7 thousand; with 80% polys, and 20% lymphocytes. No immature cells were seen. Platelets were 68.4 thousand. Normoblasts were seen on the peripheral smear; and a few microspherocytes. but not large numbers. Red cell fragility showed hemolysis beginning at 0.40% and complete at 0.30% just within normal limits. Her icterus index was 44 units. No gross hemoglobin was visible in the

serum, but it was smoky in color, suggesting the presence of methalbumin.

Sedimentation rate was 80 mm/hour (Westigren). Urine disclosed the presence of much protein, coarsely granular casts; and a few red and white cells; no obvious hemoglobinuria.

It was considered that this case was probably an acute hemolytic anemia, of the Lederer type. Her general condition was not good. Splenectomy was discussed, but it was decided to see how she did on blood transfusions, and if possible, defer the operation, or avoid it. During the next week she received daily blood transfusions of 200-500 cc. a day, which she took well, having no reactions except for the last day, when she experienced a slight chill. By May 6th she was obviously improving. Her temperature which had remained between 102-103 came down to normal over the three-day period of May 6-9th, her pulse rate decreased, and she was stronger and was hungry. Her liver and spleen, which had increased somewhat in size, began to recede. She had a bout of epistaxis on May 3rd, but no further bleeding episodes except for epistaxis on May 21st.

In the meantime other tests had been done. In order to see if hemolysis were demonstrable in her blood, her serum was mixed with washed group O cells, both her own, and those of healthy donors. These mixtures were left together overnight at room temperature, at ice-box temperature and at incubator temperature. They were set up in dilutions from ½ to 1/50. No hemolysis occurred in these tubes. No active hemolysis was therefore demonstrated in her serum by this test.

In order to see if human globulin might be adsorbed on her red cells, a sample of blood was sent away for a Coombs test. In this the effect of antihuman globulin serum is tried, and if human globulin adheres to the red cells they are agglutinated by this serum. However, the laboratory rejected the blood because of hemolysis. Her Cephalin Flocculation test was XXXX; Thymol Turbidity was 2 units; Thymol Flocculation was negative and Takata Ara was negative. Her serum proteins were 5.8 gms.%; with albumin of 3.2 gms.% and globulin 2.6 gms.%. Cold agglutinins were not present; some cases are reported in which cold agglutinins are present, and on exposure to cold, hemolysis may occur.

On May 4th the child was obviously jaundiced. Her Van DenBergh reaction done then disclosed Immediate Direct 0.60 mgs.%; Indirect 2.40 mgms.%; Total 3.0 mgs.%. The increase, therefore, was almost all in the indirect fraction, which is expected in hemolytic jaundice. With great increase in red cell destruction, it is expected that no bile appears in the urine, but increased amounts of urobilinogen.

Urobilinogen determinations of her urine showed the following results, expressed in mg. excreted per day.

May 7th	28.5 mg.
May 8th	28.5 mg.
May 9th	14 mg.
May 11th	0.2 mg.
May 12th	0.2 mg.

On May 11th the Van DenBergh reaction showed a total of 1.4 mgs.%; of which 0.28 mg. were immediate direct. Repeated red cell fragility curve at this time showed no change from before.

Abnormal lycolecithin content of blood has been reported in some cases of hemolytic anemia, and this was determined in this case, with normal findings. Her B.U.N. on May 4th was 43 mgs.%. On May 11th it was 23 mgs.%. Agglutinations for brucella, typhoid and paratyphoid were negative. Bleeding and clotting times were done May 2nd and were normal.

Other therapy the child received, in addition to blood transfusions and penicillin, was iron, vitamines including K, and liver 1 cc. daily. These were discontinued on May 24th. By May 21st liver and spleen were no longer palpable, and the child was clinically, almost recovered.

The hematological findings are tabulated:

Date	Hgb%	RBC millions	WBC thousands	Retics	
2:5:49	20%	1.1	8.7 P.80 L20	.....	normoblasts seen.
5:5:49	20%	0.93	18.4 P.41 L59	.....	normoblasts fewer.
7:5:49	36%	1.4	16.8 P.70 L27 E1	.....	few normoblasts.
9:5:49	32%	1.34	10.1 P.61 L38		
11:5:49	28%			28%	
16:5:49	36%			11%	
17:5:49	40%			1%	
19:5:49	42%			4%	
June 21st	80%	4.0	SMEAR; Normal appearance, platelets normal, in appearance.		

By the end of June the child was normal as far as clinical findings went, and her blood smear was normal in appearance. Her physician reports that she has remained so to date.

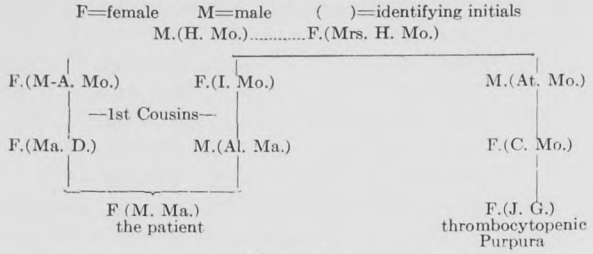
Family History

The pertinent family connections are shown in Table 1, in so far as they could be ascertained. The only other member of the family that a blood dyscrasia could be traced to, was a cousin of the patient, who died in St. Boniface Hospital in 1945, at the age of six, from subarachnoid hemorrhage, which was a complication of an acute thrombocytopenic purpura, idiopathic in type. A review of the case history of this child did not reveal any laboratory or clinical evidence of hemolytic anemia.

The blood was examined of the father and mother of the patient, and of her grandfather and grandmother. No abnormalities were discovered in these individuals.

It is seen that the parents of the patient were cousins, and this might be of some significance.

Table 1



Discussion

This case, therefore, was that of an acute idiopathic hemolytic anemia, which appears to have been a self-limited disease, and which fits the criteria of the Lederer type. It is of interest that a family history of thrombocytopenic purpura was uncovered, because the purpuric manifestations shown by this child during her illness were unusual, although they have been reported in association with Lederer's type of hemolytic anemia.

Lederer's Anemia, Brief Review

This has been defined as an acute, acquired hemolytic anemia of unknown etiology.

It was perhaps, first described by MacIntosh and Cleland[1] in 1902, and the condition was first more thoroughly investigated by Chaufford and Minkowski. In 1925 Lederer "re-discovered"[2] this type of anemia and first reported the beneficial effect of transfusions. It has since been commonly called by this name.

Incidence

It is uncommon. By 1940[2] about 100 cases had been reported, and approximately 35 further cases have been recorded between 1940 and June, 1949.

Cheney and Denenholz[9] found one case in 11,000 enlistments of military age.

Age and Sex

Most commonly seen in the first and second decade, the disease may occur at any age. Sexes are equally affected.

Diagnosis

Dameshek and Schwartz[2] in their excellent review of the acute acquired hemolytic anemias, reject many of the cases previously reported as such because of certain findings, and postulate the following criteria for diagnosis:

1. History of acute onset with rapidly progressing pallor and weakness.
2. Signs of severe anemia with icterus and splenomegaly.
3. Evidence of increased blood destruction.
4. Evidence of increased blood formation.
5. No evidence of pernicious anemia.
6. No evidence of any obvious cause of hemolysis such as bacteria, chemicals, antibiotics, neoplasm, pregnancy, etc.
7. No evidence of similar disease in the family.

The Symptoms Are

There is no specific symptomatology, but generally speaking, the symptoms fall into three groups, any of which may be present to a varying degree:

1. Those of an acute febrile illness with general malaise, fever, irritability, for a few hours to a few days.
2. Those pertaining to the gastrointestinal tract such as anorexia, nausea and vomiting, diarrhea (more commonly in infants), and abdominal pain which may be vague and generalized, or more localized to the left upper quadrant.
3. Those of anemia, with weakness, dyspnea, fatigue, and palpitation and edema of ankles.

The Signs Are

Fever (99-105°); rapid pulse (100-140); respirations tend to be increased. The sclerae are icteric and there is a yellowish tinge to the skin but the patient is "more pale than jaundiced."

Retinal hemorrhages have been reported in severe cases.

Hemic murmurs, usually systolic, are heard in a tachycardic heart.

The liver is frequently enlarged. The spleen is almost always enlarged. There may be edema of ankles. In very severe cases, uremic signs may appear (stupor and coma)[3].

The remainder of the physical examination is normal, including the tongue and nervous system.

Laboratory

Hemoglobin and red cells are reduced, the cells to 1-2 million or less with hemoglobin levels of 20-50%. The color index ranges from 0.7 to 1.4. Reticulocytes are increased to 10% or so, and may go as high as 60% with nucleated red cells present. Polychromasia is common and macroctyes (pseudomacrocytes) are characteristic, particularly in the younger age group. Spherocytes may be seen on the smear and it is in this group that the erythrocyte fragility is positive and hemolysins are demonstrated. The sedimentation rate is elevated and may be as rapid as 80 mm. in one hour.

The white cells are markedly increased to 20,000 or even 50,000 with a polymorphonuclear predominance. Many immature neutrophiles and myelocytes are commonly seen. The platelets are normal or moderately diminished. The serum icterus index averages 25-35 units, and the bilirubin levels run from 2-10 mgs.% with mainly indirect type increased. Hemolysins may be shown to be present. The marrow is hyperplastic, and shows proliferation of normoblasts.

The urine urobilinogen and urobilin is increased. Hemoglobinuria occurs where hemolysis is massive. Albumen and casts are usually seen during the acute phase. The stools usually show marked urobilin increase.

Differential Diagnosis

1. Pernicious anemia: Absence of glossitis and nervous system involvement, hydrochloric acid in

the gastric contents, and normoblastic bone marrow differentiate Lederer's from pernicious anemia.

2. Lymphoblastoma group: Absence of lymphadenopathy, and the very acute onset are helpful.

3. Acute Leucemias: These are generally differentiated on hematological grounds, but if many immature cells are present it may not be so easy. Bone marrow aspiration almost always differentiates these diseases.

4. Congenital hemolytic anemia: This may be difficult to rule out; absence of family history; absence of preceding history, and absence of typical facies or bone changes may be of help. Also the subsequent course of the disease, and examination of relatives for evidence of spherocytosis.

5. Sepsis: This may be difficult. Blood cultures, and other features of the examination must be assessed.

6. Other acute hemolytic anemias such as allergic ones (Favism, etc.), or those due to drugs and so forth, must be differentiated on history, and on other clinical evidence.

Pathogenesis-Pathology

"A body divided against itself"—such is how Lederer's anemia has been described. On the one hand we see a marrow factory booked to capacity producing life-giving erythrocytes, and on the other there is the spleen heavy with scenes of destruction of the same cells. The marrow, though hyperplastic, is normoblastic and shows none of the megaloblastic activity, nor any of the misshapen metamyelocytes as seen in pernicious anemia. The spleen is large and has congested sinusoids; there is marked proliferation of histiocytes with active erythrophagocytosis; or the picture may be mainly one of erythrostasis with many venous thrombi, and infarcts scattered throughout the spleen. The liver and kidney may show some degree of hemosiderosis at post-mortem.

Lederer's anemia is frequently referred to as a macrocytic anemia, but this is only a pseudomacrocytosis, for Dameshek and Schwartz have shown by Price-Jones curves that it is only the young, recently formed erythrocytes that are macrocytic, while the others are not.

Mason[3] agrees with Dameshek and Schwartz that the spherocytes seen in this disease are normally formed cells which have been altered in some way after entering the circulation.

This brings up the question: What precipitates this sudden destruction of erythrocytes? Spherocytosis is intimately related to increased fragility but what causes spherocytosis? Hemolysins have been demonstrated in some cases but in no means the majority.

Erythrostosis in an abnormally functioning spleen (Ham and Castle) with consequent agglutination and hemolysis does not explain why the spleen should suddenly behave so. Sturgeon[4] using serum from rabbits immunized with human serum was able to demonstrate what he terms an incomplete or "blocking" type of antibody in the red cells and free in the serum of three patients with acquired hemolytic anemia. Roelsen and Sorberge-Ohlsen[5] suggest mental stress as a precipitating factor. As yet it would appear that there is no adequate explanation. Dameshek and Miller[7] conclude there is a combination of factors brought into play in Lederer's anemia. Finally several authors believe there is a sub-acute and a chronic form of the disease. Schwartz includes an increase of lysolecithins as an etiological factor.

Treatment

1. Transfusions give dramatic results in many cases. Careful cross-matching cannot be too strongly emphasized. Some cases have shown hemolysins to some blood of O group or of their own group.

2. If transfusions fail then splenectomy is the treatment of choice. Nor is it wise to persist with transfusions if no immediate result is obtained lest the patient develop new hemolysins or agglutinins. Dameshek[8] states that no more than 3-4 transfusions should be given without result before splenectomy is considered.

3. Parenteral liver therapy and iron are of no avail.

4. Oxygen is invaluable as supportive therapy.

Prognosis

This is poor without treatment, although spontaneous recovery does occasionally occur. Transfusions effected complete recovery in 44 out of 66 cases receiving blood.[2] There remains 1/3 of the cases who go on to splenectomy. Dameshek and Schwartz[2] report 20 complete recoveries out of 23 cases who went on to splenectomy. Low erythrocyte counts do not appear to be a deterrent to splenectomy, for they operated successfully on 3 patients with R.B.C. counts of 1.3, 1.5 and 2 million respectively. The over-all mortality rate for splenectomy in cases of acute acquired hemolytic anemia refractory to transfusion is 44% (10 out of 44 cases).[8] Recovery, when it occurs, is complete and the disease does not tend to recur.

I wish to express my gratitude to Dr. Paul Green for his guidance in preparing this paper:

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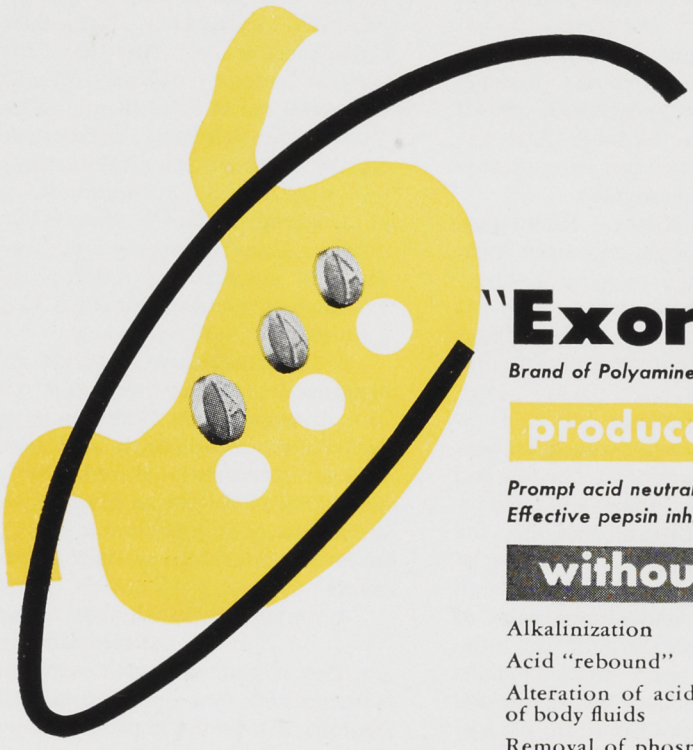

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CANCER

Edited by D. W. Penner, M.D.

Abstract

Arminski, Thomas C. (The Grace Hospital, Detroit, Mich.), Primary Carcinoma of the Gallbladder, Cancer, 2: 279-398, May, 1949.

A complete summary of the literature with addition of new cases is presented. All phases of the disease are discussed. It is difficult to evaluate the correct incidence of carcinoma of the gallbladder. The incidence varies from a 10% of all cancer deaths (Mortality Statistics) to approximately 0.36% for all autopsied. Graham has estimated that the frequency of death from primary carcinoma of the gallbladder is three times that of the lip, twice that of carcinoma of the rectum and the same as from carcinoma of the breast. The incidence in biliary tract operations is given as approximately 1%.

The age distribution corresponds to the general age groups for carcinoma with only a few cases reported prior to the age of 30. Approximately 75% occur in females.

Considering the possible etiologic factors a review of the literature shows that 75% of all carcinomas are associated with calculi, but there is no uniformity of opinion as to their relation. Cholecystitis is also uniformly found associated with carcinoma of the gallbladder. No definite conclusion can be drawn from the literature regarding the origin of gallbladder carcinoma in papillomas and adenomas. A few carcinomas unundoubtedly do arise in pre-existing benign adenomas.

There is no distinct clinical picture of primary carcinoma of the gallbladder. Approximately 50% of carcinomas are associated with a definite history of previous gallbladder disease including colic and dyspepsia. Dull pain in the upper right quadrant is present in over 50% of the cases. Progressive jaundice (painful) with an upper quadrant mass and weight loss occur in more than half the patients. Blood studies may show an anemia. Examination of gallbladder bile for tumor cells may be of value. Roentgenographic studies usually show a non-functioning gallbladder.

The gross pathology of carcinoma of the gallbladder depends upon the type and duration. The fundus is the commonest site of origin. 44% of gallbladders are enlarged and 21% are contracted and smaller than normal. In early cases the tumor may present as a thickened plaque in the wall. A common complication (about 10%) is a perforation into one of the adjoining viscera or the peritoneal cavity.

Microscopically 91% of the tumors are adenocarcinomas, 5% mixed type and 4% squamous cell type. The latter is usually considered to arise on a basis of squamous metaplasia. Approximately 20% of the tumors are papillary or villous and invasion of the wall in these tends to occur late.

Tumor spread is primarily by direct extension to the liver. Metastasis appear, first in regional nodes and liver, later to any site in the body. Even in cases designated as early, less than 20% are found to be confined to mucosa and submucosa. In grade 1 tumor 25% showed metastases at time of operation, whereas in grade 4 tumors 100% had metastasized.

The treatment of primary gallbladder carcinoma is highly unsatisfactory. In only approximately 30% is it possible to attempt a curative procedure (cholecystectomy with or without partial liver resection). Only in the cases of early low grade tumor is there a good chance for cure. The advocates of radical surgery have not obtained significantly better results. It is of interest to note that in this connection Keen, in 1899, reported 63 successful cases of partial hepatectomies.

The author's summary of 25 new cases of primary carcinomas of the gallbladder presented much the same features as described above.

In the discussion of the problem of primary gallbladder carcinoma the author disagrees with the argument for removal of all diseased gallbladders to prevent carcinoma. The author uses as a working basis the assumption that cancer of the gallbladder occurs in 3% of cholelithiasis (reported incidence varies from .63% to 30%). Calculi in females in the cancer age are very frequent—in the fifth decade the incidence is reported as 22%, the sixth decade 30%, seventh 38% and eighth 46%. This would mean a tremendous amount of surgery which the author does not believe would be justified. He agrees with Warren and Balch who state "The decision as to the advisability of cholecystectomy in cases of gall stones with few or no symptoms should be governed by a consideration of the danger of non-malignant complications rather than by risk of carcinoma of the gallbladder." The non-malignant complications listed by the author are acute cholecystitis, choledocholithiasis, pancreatitis, cholangitis and hepatitis. In various series these complications varied from 10-40%. In a 2-year follow-up of 150 cases of non-functioning gallbladders and who refused operation, 27% required subsequent operation for serious complications.

D. W. P

PATHOLOGY

Clinical Pathology (3)

Paul T. Green

Red Blood Cell Counts

Essentially, there is only one way of determining the number of red blood cells in blood, and that is to make dilutions of the blood using a diluting fluid that does not alter or destroy red cells appreciably, and then to count the number of red cells in a known volume of the diluted blood. Before discussing the procedure for determining red cells by dilution and counting method, however, it is necessary to discuss another way of determining red cells that is appearing in the literature.

Turbidity Method of Determining Red Cells

The theory behind this method is, that a suspension of red cells in the proper suspending medium will obstruct passage of light, and that the amount of light that is prevented from passing through the suspension is proportional to the concentration of red cells in the suspension. This method is mentioned only to be condemned. It is applicable to red cells that have a normal volume and hemoglobin content, but does not apply to abnormal red cells, in our experience. Therefore there is nothing to be gained from this method, because just as good an approximation could be obtained by multiplying the hemoglobin percentage by the average "100%" red cell count (approximately 5.4 millions), and in this way "determining" the red cell count. It is important to know if the red cell count has been determined by this method, so that too much attention is not paid to it.

Diluting Methods

This is the standard, acceptable method for determining red cell counts. There are, of course, inherent errors in the method, and these can be considered, as the method is described.

1. Obtaining blood sample. As pointed out before, a good sample of the blood must be obtained. This can be either finger-tip blood, or venous oxalated blood. As has been pointed out before, if much tissue juice is squeezed out while obtaining the finger-tip blood, then the blood sample obtained will be dilute, and the final value will be low.

In oxalated blood, if the blood is not well mixed before sample is removed, then a true sample will not be obtained. For example, if the blood had been standing, so that the red cells had settled out, if the technician did not resuspend the cells properly, and took her sample from the top, she would have more plasma than cells, and would obtain low red cell values; or if she took her sample from the bottom of the bottle, where the red cells had settled, she would obtain more cells than plasma, and her results would therefore, be falsely high. Now most technicians are trained well enough to watch out for this type of error. However, some bloods show marked agglutination of the red cells, and in these cases sampling from venous blood, if the cells are not properly suspended, will give erroneous results. Therefore, be sure that the sample is a proper sample.

2. Taking a measured amount of blood. Generally this is done in special red cell counting pipettes. These pipettes, as supplied by the manufacturer, are supposed to be within 3% of the stated volume. However, this is not generally guaranteed. To be certain, the pipette can be checked, by the laboratory, or else pipettes certified by the Bureau of Standards can be purchased.

As a rule, blood is drawn up to the 0.5 mark; this mark does not indicate any particular volume of blood; it is a volume that is 1/200th of the capacity of the bulb of the pipette. Diluting fluid is then drawn in, until the fluid level reaches the 101 mark. "001" is there in order to allow for the diluting fluid that fills the stem of the pipette, and which does not enter the bulb, and therefore does not figure into the dilution of the blood. This volume is discarded eventually.

The pipette, of course, must be clean and dry. There must be no bubbles of air in the column of blood, and the volumes must be measured as accurately as possible.

3. Diluting Fluid. The fluid used for dilution should not destroy red cells, nor should it clump them. Solutions used in the past have had a tendency to produce clumping of some red cells (for example Hayem's solution). When red cells are clumped they cannot be shaken into a uniform suspension throughout the diluting fluid, and they tend to stay in clumps on the counting chamber, so that two or three red cells may be counted as only one red cell, and hence produce erroneously low red cell counts.

Satisfactory diluting fluid is made as follows:

Sodium Citrate	3 gm.
Water up to	100 ml.
Formalin	1 ml.

Filter. Keep in refrigerator.

4. Dispersing the Cells. Once diluted it is necessary to disperse the red cells uniformly throughout the fluid. This can be done by shaking manually. It takes several hundred good shakes to disperse the red cells evenly. Shakers are on the market for doing this now; a vibrator type is comparatively inexpensive, and is quite efficient. A few minutes on this shaker will produce a good dispersion of the red cells.

5. Counting chamber. A portion of the fluid is now to be taken, so that the red cells can be counted. This counting is done in a counting chamber, which is a glass chamber so designed that when the glass coverslip is in place, there is a depth between the cover and the base of 0.1 mm. (according to Bureau of Standards specifications, this depth must be 0.1 mm. within 1% error); and the central square is divided into 25 smaller squares, whose edges are double or triple ruled to identify them. Each of these squares is, therefore, 1/25 mm. square, and 1/10 mm. deep, and therefore has a volume of 1/250 cubic mm.

The diluted blood is in the pipette, and the red cells have been evenly dispersed throughout the medium. A portion of the fluid is blown out and discarded, in order to get rid of the diluting fluid in the stem of the pipette which does not contain red cells and does not enter into the diluting (the "001" fraction mentioned above). A portion of the remaining fluid is then allowed to run into the chamber; this must be done carefully so that the chamber is completely filled, but does not overflow; if it overflows the cover slip will float up and the volume will be greater than it should be. Also surface tension draws red cells out to the sides and a true dispersion of red cells does not occur. The chamber is now allowed to sit for five minutes or so, so that the red cells will settle to the bottom. The chamber is now surveyed under low power, in order to be certain that the red cells are evenly dispersed over the chamber. The red cells in five of the 1/250th cu. mm. areas are now counted under high power—usually the four corner ones and one central one—and the number in each of these squares should be close to each other. If they are not, then the red cells are not evenly dispersed over the chamber. The problem of what cells to include in each square arises because some cells will be on the boundary line of the area. This is overcome by including into the square all red cells on the upper or "north" boundary line, and the left or "west" boundary, and excluding those on the "south" and "east" boundary lines.

Calculation

Each square counted contains the red cells present in 1/250th cubic millimeter of diluted blood. Five of these were counted, or a total of 5/250th cubic millimeters. Therefore, if X be the total number of red cells counted in the five squares, there are 250X/5 red cells in each cubic millimeter of diluted blood. Our dilution was 1/200th. Therefore the number of red cells in each cubic millimeter of blood originally taken is:

250X/5 times 200, or "X times 10,000."

Error in Determination

It is agreed that the error in determining red cells is considerable. One group has found that the errors in pipettes and counting chambers runs about 4%; that observer error runs about 3%, and that random error can be as high as 7-8%. They found that the total error in doing routine red cell counts was something less than 10%.

Other observers have found similar values. If one does duplicate determinations on a group of patients by taking one red cell count from one finger of one hand and another count from the same finger on the other hand, standard deviation from the mean runs .39 in hundreds of thousands, about 8% (if duplicates are done and the average of the duplicate determinations is used, this standard deviation is reduced to 0.28 hundreds of thousands).

This means, in effect, that red cell counts done on the same individual must differ by 10% or more, before one can be certain the count represents a true difference.

For example, if the red cell count in a patient is found to be 4.21 millions; and he is given liver injections, and two weeks later his red cell count is 4.4 millions, this does not represent a significant difference, and one could not say that his red cell count had increased, as these difference are well within the error of measurement of red cells.

Normal Values

In males, the average red cell count is 5.4 millions. 95% of normal males fall within a range of 0.8 to either side; or "normal" red cell count for males is between 4.6-6.2 millions.

For females, the range is 4.2-5.4 millions.

Significance of Abnormal Values

Red cell counts, like hemoglobin values, are not measurement of total body red cells or hemoglobin, and therefore can be falsely high or low because of hemoconcentration or dilution, which should be considered in assessing reported values.

Abnormal red cell counts must be regarded as a sign of possible disease, and calls for evaluation of clinical and other laboratory tests. It has no diagnostic significance as an isolated finding.

Use of Red Cell Counts to Standardize Hemoglobinometers

Hemoglobinometers can be calibrated fairly well using cell counts to do this:

Venous, oxalated blood is obtained from three healthy male donors. Red cell counts are done on each of these bloods, in triplicate. The three counts on the same blood should check within 10% of each other and, of course, they should be within the normal range.

Average the red cell counts, so that an average red cell count for each of the three bloods is obtained.

Calculate the hemoglobin percentage for each blood as follows:

$$\frac{\text{Red cell count} \times 100\%}{5.4 \text{ millions}}$$

Assuming that this is the correct hemoglobin value for each blood, calibration curves can be made up by making various dilutions of each of the bloods, and proceeding to determine its hemoglobin by whatever method is being used, and in this way to derive a calibration for the method.

Example, using only one blood:

Red cell counts: 5.2 4.9 5.4 average = 5.2

Assumed % hemoglobin is $\frac{5.2 \times 100\%}{5.4} = 95\%$.

Into a series of four tubes, place the following amounts of saline in serial order:

Tube 1	0 cc.
Tube 2	0.5 cc.
Tube 3	1.0 cc.
Tube 4	1.5 cc.

To each tube add the following amounts of well mixed blood:

Tube 1	2 cc.
Tube 2	1.5 cc.
Tube 3	1.0 cc.
Tube 4	0.5 cc.

The presumed hemoglobin % in each of these tubes, then is:

Tube 1	95%
Tube 2	72%
Tube 3	48%
Tube 4	24%

Into a series of colorimeter tubes place 4 ml. of 0.1% Sodium carbonate solution. To the first tube add nothing. To the second tube add 0.02 ml. of blood from Tube 1, and repeat for each subsequent tube. Mix well. Using the tube containing carbonate only adjust the colorimeter to read 100% transmission, using a green filter. Read the values for the other four tubes.

Plot on semilogarithmic paper, the results. These readings should be on a straight line that passes through zero concentration at 100% transmission. This now gives a curve from which hemoglobin values can be read off, when the transmission is known.

The other bloods should give readings which fall close to this line. When using several bloods, the best line is run through the points.

Clinico-Pathological Conference

Medical Department, Deer Lodge Hospital

Dissecting Aortic Aneurysm

M. Cohen, M.D.

Mr. S. K. Born Jan. 1, 1885. Occupation, odd jobs on farm.

April, 1915—Wounded in right anterior chest while on active service. No operation required. No past illnesses noted. Discharged in 1919 as physically fit.

1919-1947—Worked in rural Manitoba at odd jobs—mostly physical in nature. General health good—no illnesses except for occasional cold. Family history non-contributory — one brother killed in action. Two brothers alive and well. No history of T.B., Ca., Diabetes, nephritis, heart or blood disorders.

April 19, 1947—At 5 a.m. was awakened by a severe sharp pain in the left foot radiating up the entire limb to the groin—thence to the anterior abdomen below the umbilicus, and around both loins to the kidney areas. The pain was severe, knife-like and made the patient break out into a sweat and clench his teeth. At the same time the left leg and foot felt numb to touch and the limb went limp. The severe pain lasted 15 to 20 minutes and then eased considerably. Pt. could then get out of bed and walk, but the leg was still numb and he limped. Walking short distances did not aggravate the pain.

2 p.m. Seen by physician who noted diminished left pedal pulses—advised admission.

8 p.m. Admitted to D.L.H. On admission—one examiner noted a coarse systolic thrill in the abdomen just below and to the right of the umbilicus. The left foot felt cool but is of the same temp. as the right. Left dorsalis pedis, post tibial and popliteal pulses not palpable. Capillary return in toes good. Left femoral pulse palpable but not as full as right. BP over right popliteal space 130/60. BP over left not obtainable. No areas of anaesthesia or discoloration. Movements normal.

April 20, 1947—Seen on Ward. Review of system essentially normal.

Physical Examination—Elderly gentleman, in no apparent distress, slightly deaf and tends to minimize his complaints.

Head and Neck—Retinal vessels show irregularity and nicking, otherwise negative.

Chest—Barrel-shaped, asthenic. Negative to I.P.P. & A. Movements restricted moderately—heart—apex 5 I.C. 4" from sternum. BP 135/80. Rate 52, regular; a harsh systolic murmur is heard over apical area radiating to the aortic area.

Abdomen—Some tenderness in L.L.Q. Otherwise negative. Rectal—negative. Above noted thrill is not felt by this examiner.

Extremities—Feet are cool, pale. Left femoral pulse faint but definite. Pedal pulses not felt.

April 21, 1947—X-ray reports: Chest—"The aortic shadow is slightly broadened and elongated —otherwise negative."

Abdomen—"A number of small opaque, metal fragments are seen overlying the right upper abdomen. Fairly well marked hypertrophic changes are seen in the lumbar spine. The right kidney outline appears normal. The left is obscured by gas shadows. No calcification is seen in the aorta in the A-P view."

Blood Examination and Urinalysis — within normal limits.

April 22, 1947—Seen on ward—no change noted. In passing mentioned to his doctor that he had some slight precordial pain that morning. He died very suddenly in 15 minutes without disturbing the patients in the surrounding beds.

Autopsy Reports

General Description—The body is that of an elderly male 5 feet 7 inches in length. The pupils are equal and measure 4 mm in diameter—a well marked arcus senilis is present. The left leg and thigh are pale and there is a bluish discoloration over the left thigh 3 cms below and just lateral to the mid point of the inguinal ligament.

Cranial Cavity—The brain weighs 1370 gms—no pathology is seen.

Thoracic Cavity—The pericardial cavity is distended with blood which on aspiration measured 625 cc (partially clotted). A tear is present in the wall of the aorta and on the anterior surface just opposite the tear in the aorta a defect is seen in the pericardium which is the point of entry of the blood. The chambers and valves are all within normal limits. The aortic valve has calcium in the base of each cusp—the commissures are normal. There is a transverse tear in the aorta 15 mm above the valve cups and including most of the circumference of the aorta. The media is dissected up to the great vessels and down to the level of the valve ring, being ruptured into the pericardium just at the right margin of the heart where the aorta and right atrium come together. There is some atheroma in the aorta but it is not exceptional.

The left common iliac artery is occluded by blood clot in the wall of the artery which completely occludes the common iliac and extends for 1 cm into the external iliac. There is no evidence of syphilis of the aorta.

The right pleural cavity contains 140 cc of blood tinged fluid. The lung weighs 450 gms. The lower lobe is dark, congested and edematous—otherwise nothing of note.

The left pleural space contains 70 cc of similar appearing fluid. The lower lobe is congested.

Abdominal Cavity—A few cc's of blood tinged fluid are present in the pelvis.

The gastro intestinal tract is normal.

The liver weighs 1730 gms and is grossly normal except for a mild nutmeg appearance.

The spleen, pancreas, kidneys and adrenals showed nothing of note.

The thyroid, bladder and prostate were normal.

Summary

1. Hemopericardium due to rupture of dissecting aortic aneurysm—due to
2. Medionecrosis of the aorta.
3. Obstruction of left common iliac artery due to dissecting aneurysm.

Microscopic

Arch of Aorta—Section shows areas of necrosis in the media with splitting of the media by hemorrhage. The adventitia is hemorrhagic and shows newly formed fibrous tissue growing out into the blood clot lying between the media and adventitia. There is some scattered round cell infiltration.

Left Ventricle—Section shows some fragmentation of muscle fibres.

Lungs—Some hyperemia of the lower lobes.

Liver—Some parenchymatous degeneration of the liver cords.

Left common iliac artery—Shows dissection in outer part of the media—with some medionecrosis.

Dissecting Aneurysms

Definition—This is a hematoma of an arterial wall in which blood passes up and down in the middle coat of a large artery, nearly always the aorta, separating that coat into two layers. It is not a true aneurysm because that vessel is not dilated.

Etiology—It is essentially a weakness in the media—in the aorta it is a medial necrosis of unknown cause (Erdheim 1929). A second factor usually of great importance is hypertension; a third factor infrequently is atherosclerosis of the arterial wall. Syphilis is very rarely a contributing cause (3%) (Mate and Carr, 1942).

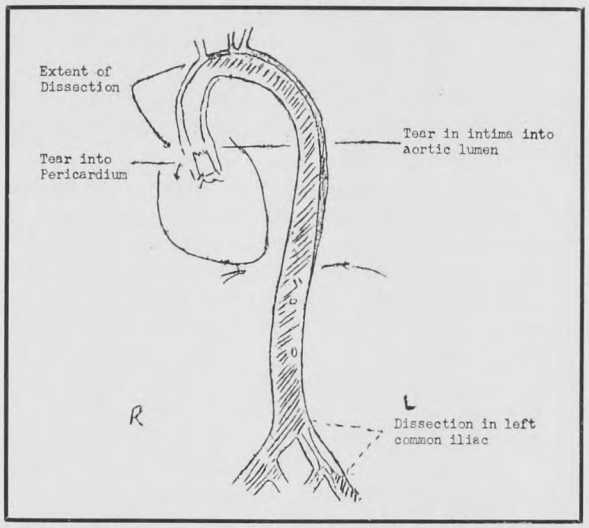
Age Incidence—In middle aged and elderly most commonly—rarely before age of 30.

Sex—Males 3 to 1.

Occurrence—Once in every 200-552 necropsies. Of 300 to 400 reported cases all but a few were in the aorta.

Pathology—These involve chiefly the aorta, occasionally the first portion of its larger branches due to extension and rarely other vessels such as the coronary arteries independently. Dissection begins in the media as a result of rupture of the vaso-vasorum, but the intima quickly breaks through into the medial lesion by tearing sharply in a horizontal or oblique direction, part way around the inner circumference of the aorta in its ascending portion or in the arch, less commonly in the descending portion, in the thorax or abdomen. Under a high head of pressure (hypertension) the intra aortic blood penetrates into the media and as a rule splits it up and down and

occasionally dissects its entire length from aortic valve to bifurcation of the common iliac. The dissection occurs ½ to ¾ of the circumference of the aorta and the blood in the medial split bulges the wall out in a variable but usually only moderate extent of 0.5 to 1 cm. Secondary tears through the intima are likely to be found at the upper and lower ends of the dissection even in one of the iliac arteries—thus producing an extra channel through which blood passes. In most cases in the course of minutes, hours, or days, the aorta ruptures completely as a result of a tear through the adventitia with sudden death due to hemorrhage into the pericardial or pleural cavity; in a few cases the lesion heals sufficiently so that the extra-aortic channel becomes lined with endothelium to give a double barrelled aorta. A constant and im-



portant complication is the involvement in the process of dissection of the mouths of the aortic branches and resulting effects of blocking of local circulation as in the case of an iliac, or coronary, or intercostal artery. Careful search of the aortic wall histologically at the site of dissection has revealed in most cases an area of unexplained degeneration or necrosis in the media. This may involve predominantly the muscle cells and connective tissue or the elastic lamellae and collagen fibers (medionecrosis aortae idiopathica cystica of Erdheim, 1930). Many ruptures of the aorta have the same pathology as dissecting aneurysms but the adventitia has ruptured immediately and death supervened with no dissection.

Fifty per cent rupture into the pericardium, the rest into the left pleural cavity, mediastinum, retroperitoneal tissues or elsewhere. External ballooning may compress neighboring structures—a remarkable example being of George II, whose widely dissecting aneurysm compressed the pulmonary artery with resulting rupture of the right ventricle—the catastrophe occurring while straining at stool. 4% occur in advanced pregnancy.

Symptoms—In a few cases the arterial dissection may be apparently symptomless or so obscure in symptomatology that it cannot be diagnosed. As a rule, however, there are 2 symptoms that are more or less characteristic which when present together are almost pathognomonic. (1) Pain attending the splitting of the aortic wall is usually excruciating and extensive radiating from the mid thorax through the chest, down the back and even into the thighs or up into the neck. The pain in the thorax or back comes on suddenly at its maximum and is often prostrating, inducing shock, or even death. If the patient survives the pain usually lasts for hours (sometimes 24-48 hours) requiring morphine repeatedly. It may recur if there is an extension of dissection. (2) The other symptom or group of symptoms is dependent on the blocking of the circulation to some important part or parts of the body, especially legs, viscera, or brain. Pain, numbness, coma, and other symptoms may result.

Signs—None are pathognomonic. In the case of the aorta, a systolic murmur at the base of the heart, transmitted to the neck and along the spine may be heard, and an aortic diastolic may be heard, but these are far from constant. Chronic hypertension and some cardiac enlargement therefore, are almost invariably present. The blood pressure often remains high after dissection, or it may drop during the initial shock and then rise again with recovery. Arterial pulse obliteration may be found due to compression of one of the aortic branches—seen in the legs more commonly along with the other signs of vascular occlusion.

Fever and leucocytosis of slight to moderate degree are common for a few days or of low grade even for a few weeks.

X-ray examination is of little help.

E.K.G. is helpful because of its negative finding except rarely where one or other of both coronary arteries may be involved with occlusion.

Course and Prognosis—The condition is usually fatal as a result of rupture through the adventitia in the course of minutes to days in 75% to 80% of cases. Death is sudden. Certain cases may survive months to a few years and die non cardiovascular deaths. 90% die from rupture in the first few hours or days. Osler describes a man who lived comfortably for over 30 years with a double tubed aorta.

Complications—Chief ones are external rupture or blocking of the circulation to some part of the body (especially legs, heart and brain).

Treatment—Absolute rest for 6 weeks, special nursing care at the onset and symptomatic therapy by morphine for pain, shock, and symptoms of occlusion of peripheral or visceral arteries. As yet non-surgical.

Differential Diagnosis — 1. Coronary Thrombosis—Sudden onset of maximal pain instead of building up in a few minutes; secondly the con-

stant radiation to the back or often its origin there; thirdly, evidence of rapid peripheral blocking before embolism from endocardial infarction is possible; and lastly, a normal or unchanged E. K. G.

2. Blocking of Peripheral Arteries (Embolism)—often initial chest pain just prior to the arterial block (a few minutes). Absence of any adequate explanation for intracardiac thrombosis where an embolus might originate.

3. Pulmonary Embolism—is less likely to cause confusion because of the preponderant dyspnea, cyanosis and tachycardia, much less pain, history of operation or injury, occasional blood spitting and physical chest signs.

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Medico-Historical

J. C. Hossack, M.D., C.M. (Man.)

A Historical Sketch of Anatomy as a Basis for Medicine*

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One of the great events of history was an eclipse of the sun seen on the shores of the Eastern Mediterranean in the year 585 B.C. What was important about that particular eclipse was that it had been predicted by Thales, an Ionian merchant and scientist. The spectacular verification of this prediction helped to influence the early Greeks to think that perhaps after all, the diverse affairs of this universe, including the ills that flesh is heir to, may not be decided by the incomprehensibly human whims and foibles of the gods, but may be determined by orderly relationships of cause and effect, and hence may come somewhat within the scope of human understanding based upon systematic observation and logical reasoning. The gradual development of this, the modern scientific viewpoint, was one of the intellectual triumphs of ancient Greece. In this connection we venerate especially Hippocrates (about 460-370 B.C.), the father not only of Medicine, but of the scientific method of studying all natural phenomena by observation and reasoning; for clinical medicine was the first subject to be so studied systematically and extensively. Of all that was fine and splendid in "the clear, cool air of Athens," of all that was rare and beautiful, of all that was high and noble, nothing has come down to us that surpasses the Hippocratic writings in widespread and permanent value to mankind. If there be any among us in whom so glorious a heritage stirs no feelings of loyalty and responsibility, such a one has entered Medicine by mistake.

Ancient Anatomy

As already mentioned, the Hippocratic writings (which were not all written by Hippocrates) are chiefly clinical; the only anatomy that amounts to anything describes the bones and joints in connection with fractures and dislocations, together with a few other scraps, some of which concern the heart. How Hippocrates acquired his anatomical knowledge we can only guess; we have no record of human dissection in ancient Greece, and with slight exceptions ancient Greek Medicine had little anatomical basis.

Aristotle was the tutor of Alexander the Great, who in 332 B.C. founded the city of Alexandria, at the mouth of the Nile. There, under the Ptolemaic Dynasty, flourished the first great school of Science and Medicine whereof we have record. Greek in origin, it was at its best from about 300 to 100 B.C., though it lingered in decay until about 400 A.D. This was not a separate medical school, but part of a great scientific institution, built around a museum and a library, where the doctors matched wits with scientists of the calibre of Archimedes, Aristarchus and Euclid. Under such favourable circumstances, Medicine was for the first time placed on a scientific basis; this included Anatomy, which, as with everything during the best days of Alexandria, was studied scientifically, i.e., at first hand. Hence, so far as we know, it was at Alexandria that Anatomy was first cultivated as a basis for Medicine, and it was there that human dissection was first practised systematically for medical study. Herophilus and Erasistratus were two outstanding Alexandrian anatomists honoured today as the fathers of Human Anatomy.

The last distinguished medical man associated with the Alexandrian School was Galen (130-200 A.D.), who studied there when human dissection had been superseded by that of monkeys and pigs, but human skeletons, dating from better days, were still available for study. By this time the Roman Empire was flourishing, and Galen became a fashionable physician in Rome, his patients including the Emperor Marcus Aurelius. Like John Hunter, some sixteen centuries later, Galen took time from his practice to dissect animals and experiment upon them; he was thus the father of Experimental Anatomy, the study of **living** struc-

* Presented to the Second Year Class, University of Manitoba Medical College, January 6, 1950.

tures, which he established as a vital part of the basis of Medicine. He also established planned, systematic experimentation as an important method of scientific investigation. For Anatomy as a basis for Medicine, the death of Galen in 200 A.D. marked the beginning of the Dark Ages.

Renascence Anatomy

Though there had been dissecting during the previous two centuries, the Scientific Renascence came in 1543, with the almost simultaneous publication of the great work of Copernicus, "De Revolutionibus Orbium Coelestium," and the even greater work of Vesalius, "De Humani Corporis Fabrica." Andreas Vesalius (1514-64) was then only twenty-eight years of age, and was professor of Anatomy at Padua. Containing no great individual discovery, Vesalius's book is for scientists probably the most important ever written; it restored to Science the fundamental principle that observation (not hearsay) is the only true basis of scientific knowledge. Originally established by Hippocrates, this principle disappeared during the thirteen centuries between Galen and Vesalius; we cherish it today, in unbroken succession from Vesalius, as the very bedrock not only of all our Medicine, but of all our Science.

If we except the two or three golden centuries of Alexandria, only during the last four centuries has there been any reliable anatomical basis for Medicine. On that basis, the progress of Medicine has been uninterrupted from Vesalius's day to ours. Let us examine this last statement. To what extent may the progress of Medicine be fairly attributed to Anatomy? This progress has been based, more than anything else, upon observation. From a historical viewpoint, at least, it should not be forgotten that for three hundred years, from the middle of the sixteenth century to the middle of the nineteenth, in most medical schools Anatomy was the only subject in which the future doctor was adequately trained in the method of systematic observation. Even today this is the true importance of Anatomy in medical education—not so much the clinical value of anatomical knowledge as such, but rather the influence of the method by which that knowledge is acquired. Of course in modern medical schools all subjects are taught in varying degrees by the method of observation in laboratory and ward; and Anatomy can now share this burden with other subjects basic to modern Medicine. In so far, then, as Anatomy has kept the torch of observation burning, and has handed it on to generation after generation of doctors, it may fairly be said to have formed the basis of all medical progress since the sixteenth century.

Surgical Anatomy

The importance of accurate anatomical knowledge as a basis for Surgery is witnessed by its successful application at the hands of such post-Vesalian surgeons as William Cheselden, John Hunter, John Bell (brother of Sir Charles), and a host of moderns. This matter received a tremendous impetus from the development of surgery in the second half of the nineteenth century, made possible by the introduction of anaesthetics and antiseptics. Although this surgical expansion enhanced the importance attached to Anatomy, it was not an unmixed blessing, for it tended to restrict attention to that aspect of Anatomy useful to surgeons, especially when operating, namely, descriptive anatomy. This was apt to degenerate into the scholastic study of dead structures, and the memorising of descriptive formulae of mediaeval artificiality and complexity, leaving no time for students to understand what doctors are really concerned with, that is, people before they have died. So much did this descriptive aspect of Anatomy come to predominate that many doctors still cannot see that any other aspect of Anatomy possesses medical significance — indeed, to some Anatomy has no other aspects!

Pathological Anatomy

But there is a broader way in which the anatomy of the dead has formed a basis for medical progress. The accurate post-Vesalian analysis of the body into parts and organs suggested the localization of various disorders in certain anatomical structures. This idea was placed on a sound observational basis by G. B. Morgagni (1682-1771), holder of the professorship of Anatomy at Padua, a chair made famous by Vesalius two centuries earlier. In 1761 he published his "De Sedibus et Causis Morborum per Anatomen Indagatis," in which he showed that certain disease processes may be localized in anatomical structures (heart, lungs, stomach, brain and so forth), thereby laying the scientific foundation of pathological anatomy.

Morgagni's work was extended and refined at the end of the 18th century by a brilliant young Frenchman, M. F. X. Bichât (1771-1802), whose "Anatomie Générale" appeared the year before his lamentable death at the age of thirty-one. In Anatomy Bichât analyzed the organs into tissues: in Physiology he referred the functions of the organs to the properties of their constituent tissues; and in Pathology he assigned the disorders of organs to lesions affecting primarily one or more of the tissues composing them.

In 1839 Schleiden and Schwann established the cell as the unit of anatomical structure; and two years later Jakob Henle (1809-85), of renal tubular fame, published his "Allgemeine Anatomie," in which for the first time the tissues were systematically described in cellular terms. In 1858 Rudolf Virchow (1821-1902), the famous German patholo-

gist, published his "Cellularpathologie," establishing the cell as the unit of pathological change.

It is abundantly clear that the work of this apostolic succession, Morgagni, Bichât and Virchow, in progressively improving the precision of our knowledge of disease processes, was based throughout upon progress in the anatomical analysis of the human body.

Until the nineteenth century, the differences between health and disease, between the normal and the abnormal, seemed so clear and distinct that they alone were considered, and similarities were ignored. Observing that diseased tissues could be analyzed into the same anatomical units as the normal (cells, intercellular matrix, and so forth), Henle was led to the important conclusion that disease is merely the normal response of a normally working mechanism to abnormal stimulation—the natural effect of a noxious agent. This revolutionary idea, based on anatomical study (Henle was professor of Anatomy successively at Zurich, Heidelberg and Gottingen), was supported on the same grounds by John Goodsir (1814-67), the distinguished professor of Anatomy at Edinburgh, and was established by Virchow. I trust that I have made clear the part played by Anatomy as the basis of improved understanding of disease. Even this, however, was based on the study of dead structures.

Dynamic Anatomy

As previously mentioned, the pioneer in the study of living anatomical structures was Galen, the father of Experimental Anatomy. There was no great experimenter in any science between Galen and Galileo (1564-1642), who taught physics at Padua while a young Englishman named William Harvey (1578-1657) was a student there at the end of the 16th century. Harvey studied Anatomy under Fabricius (1537-1610), the successor to Fallopius in the chair formerly adorned by Vesalius. The combined influences of Galileo and Fabricius led Harvey, after his return to England, to make a remarkable series of experimental observations whereby he established the circulation of the blood. His immortal "Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus," published in 1628, not only contained the observational basis of one of the greatest discoveries ever made, but also re-introduced into Anatomy the experimental method of Galen. The overwhelming importance of Harvey's work as a basis for all subsequent medical progress is obvious; we recall that he was lecturer on Anatomy and Surgery to the Royal College of Physicians of London; and we observe that he entitled his great experimental work on the circulation an anatomical treatise—"Exercitatio Anatomica."

Our next great student of dynamic anatomy was John Hunter (1728-93), the Scottish-born surgeon who gained fame and fortune in London during the second half of the 18th century. How Anatomy served as the basis of surgical advance in his hands is well illustrated by his famous case of aneurysm of the popliteal artery. Becoming interested in the blood vessels of the velvet of the deer's antlers, he tied the artery to the velvet, and observed that after a few days collateral vessels developed sufficiently to restore normal circulation. Shortly after this he had a patient with an aneurysm of the popliteal artery that challenged his skill in treatment. His experience with the antlers suggested that if he ligated the vessel proximal to the aneurysm, the reduction of blood pressure within the sac might arrest its growth, while the arterial anastomoses about the knee developed sufficiently to nourish the leg below the aneurysm. To ligate proximal to the aneurysm he had to tie the femoral artery in the fascial canal deep to the sartorius muscle. He was rewarded with success; and for many years the sub-sartorial canal was called Hunter's canal. Many more instances could be related of how Hunter utilized his anatomical observations and experiments as a basis for advancing surgery.

As we have seen, the idea that the phenomena of disease are produced by the same bodily (and mental) mechanisms that operate in health first arose from the study of dead anatomical structures. Today, however, we look for elucidation of disease rather to the study of living things: living people by various means (anthropometry, X-rays, and numerous physiological, biochemical, and psychological tests); living organs by organ-culture, introduced by Carrel and Lindbergh; and living tissues and cells by tissue-culture, introduced by an American anatomist, Ross Harrison, for the solution of a purely anatomical problem, the mode of development of nerve fibres. Living anatomical structures, both developing and adult, are also studied extensively by transplantation experiments, and by exposure to various agencies, such as X-rays and other radiations, and certain chemicals, especially oestrogenic and carcinogenic substances, hormones and vitamins, or by the withdrawal of these. Significant of the modern trend is the fact that vitamin E, or alpha-tocopherol, was discovered by a professor of Anatomy, H. M. Evans of the University of California, who was led to it by his embryological investigations. But, you say, surely an anatomist who makes an important discovery in the field of biochemistry is guilty of trespassing. We have here one of the best features of the modern study of living things, namely, the breakdown of academic barriers, and the union of all by the bonds of a common interest. The days are past when anatomists and pathologists studied only the dead, leaving the living to physiologists and clinicians. The problems of health and disease are

problems of living people, and must be so studied by all concerned if their solution is to be hastened.

Let me invite your attention to a classical example of modern anatomical work as a basis for progress in Medicine. It had been observed from antiquity that whereas most animals have distinct breeding seasons, man breeds the year round. Moreover, women menstruate, whereas most animals do not. From antiquity to the beginning of the present century, that summarized the scientific knowledge available to gynaecologists as the basis for understanding female pelvic disorders. In the 1890's a distinguished Cambridge zoologist, Walter Heape, made for the first time a comparative study of what he called the oestrus cycle in various animals. The anatomists took told of this problem in 1915, when Stockard and Papanicolau of Cornell published their epoch-making study of the oestrus cycle in the guinea pig, correlating the anatomical changes in uterus and ovaries with the various stages in the cycle of sexual behaviour. An anatomical cycle of follicular, luteal, endometrial and vaginal changes was shown to underlie the behavioural cycle. This was confirmed for the rat by Long and Evans in California. From that pioneer anatomical work has flowed a veritable torrent of discoveries, resulting in much clarification of the menstrual cycle, and the recognition of the underlying cyclical hormonal mechanism. This in turn has illuminated many obscure female pelvic disorders, especially those associated with the menopause, and has brought many of them under welcome amelioration. The clinical study of these disorders had been going on ever since the dawn of Medicine; but more progress has been made in this field in the past thirty years than in the previous thirty centuries. This modern advance started with the anatomical observations of Stockard and of Evans; and Papanicolau has extended part of his work (vaginal smears) directly into the field of gynaecology. I can think of no recent advance in Medicine that has had a clearer anatomical basis than this.

"Theoretical" and "Practical"

Ladies and gentlemen, you are now in the transition zone between pre-clinical and clinical studies, between the study of the so-called normal and that of the abnormal. Medical students are naturally apt to be impatient to complete this transition, to abandon the "theoretical" study of good health, for the supposedly more "practical" study of ill-health. Now what would you think of a meteorologist who concerned himself only with bad weather because, he said, it is of more practical use than fine weather? Medicine will improve as the understanding and preservation of "mens sana in corpore sano" comes to be regarded as not less practical than the treatment of sick and injured people. This will be facilitated by renewed appreciation of the doctrine of Henle, Goodsir and Virchow, the doctrine of the identity of the basic mechanisms common to normal and to abnormal phenomena alike. Does a theoretical thermostat keep our temperature normal whilst another and more practical mechanism makes us feverish? Does a theoretical barostat keep our blood pressure within the bounds of normality, while it takes a more practical device to give us hypertension? Is the genetic mechanism concerned with haemophilia more practical than that determining a normal sex-linked character, such as the "breaking" of a youth's voice, or the sprouting of his beard? You are presently studying the various pathological degenerations. But is all degeneration abnormal? What about the spectacular degeneration of the entire pronephros and most of the mesonephros in every normal person, and the equally dramatic degeneration of the Wolffian ducts in females and of most of the Müllerian ducts in males? Is the healing of wounds restricted to surgery? What about the healing of the long gash between the right and left neural folds as they close and join to form the neural tube and its covering of skin and other structures? Or the union by first intention of the various elements that go to make the upper lip, the palate, and the septa of the heart? The healing of traumatic injuries is merely a modification of the fundamental healing process that manifested itself normally in each of us "when we were very young"—the ever fascinating "vis medicatrix Naturae."

But I must stop. I trust that I have succeeded not only in reminding you that there has been an anatomical basis of Medicine in bygone years, but in suggesting that the study of the problems of modern Anatomy bids fair to contribute to the basis of better Medicine in the future, and in warning you against supposing that Anatomy may safely be forgotten as soon as you enter upon clinical studies.

BOOK REVIEWS

The Physician's Business is a book about the business end of practice—that part of practice which beginners find more difficult than diagnosis. Some doctors start out with a business instinct or a business training. Even they could read this book with profit but for the others it is a necessity. It covers everything from how to pick a place to practice and how to get started, to how to invest your money when you have made it.

A good deal of attention is paid to fees and quite naturally because if these aren't collected there wouldn't be much chance to use the rest of the book. And so we find included a chart of an illness with spots marking the psychological times to bring up the question of payment. And, seeing that a lot of people never respond to such stimuli, there is a series of letters for the delinquents. These begin with the usual euphemistic "I'm sure you've overlooked this" routine and end up with a real punch. One of my young friends who bought an earlier addition assures me that these letters really work.

However, the book is not all about money. It has a section on records in which many forms are illustrated and in which guidance is given in their upkeep and filing. There is also a useful section headed "Instructions to Patients." These instructions deal with a lot of common things and are meant to be copied and given to the patient for guidance. Included are some pages of exercises for various conditions. The diagrams are so simple that they could easily be copied or traced on the patient's instruction sheet.

A tip-top section is headed Office Personnel. It tells the doctor how to treat his employees so as to win their loyalty and it tells the employees how to help the doctor and his patients. In dealing with the secretary it sets forth her duties and also tips her off to a few good ideas including how to modify the doctor's bark into a soothing coo. For example: Secretary to Doctor, "Miss Anxious is on the telephone. Can you see her?" Doctor, "That woman! No I can't see her." Secretary to Patient, "The doctor says he is sorry but he can't see you today, every minute is filled." And so the patient, if not quite satisfied, is at least kept from going elsewhere.

The duties of the nurse-assistant are given so simply and fully that, with the book to guide her, any intelligent girl could give almost professional assistance.

Ethics, medical journalism and public speaking precede the discussion of the Doctor and the Law. After this comes money again—this time investments, insurance and retirement planning. The final section deals with Social Medicine.

A certain amount of the information given does not apply in Canada but the bulk of it does. It covers all the economic problems that face the doctor from residency to retirement. There is, however, no mention about hobbies or leisure. But after all this look is on business, and if it be true that "all work and no play makes Jack a dull boy" it may be equally true that "all work and no play makes jack"—which is the chief topic here.

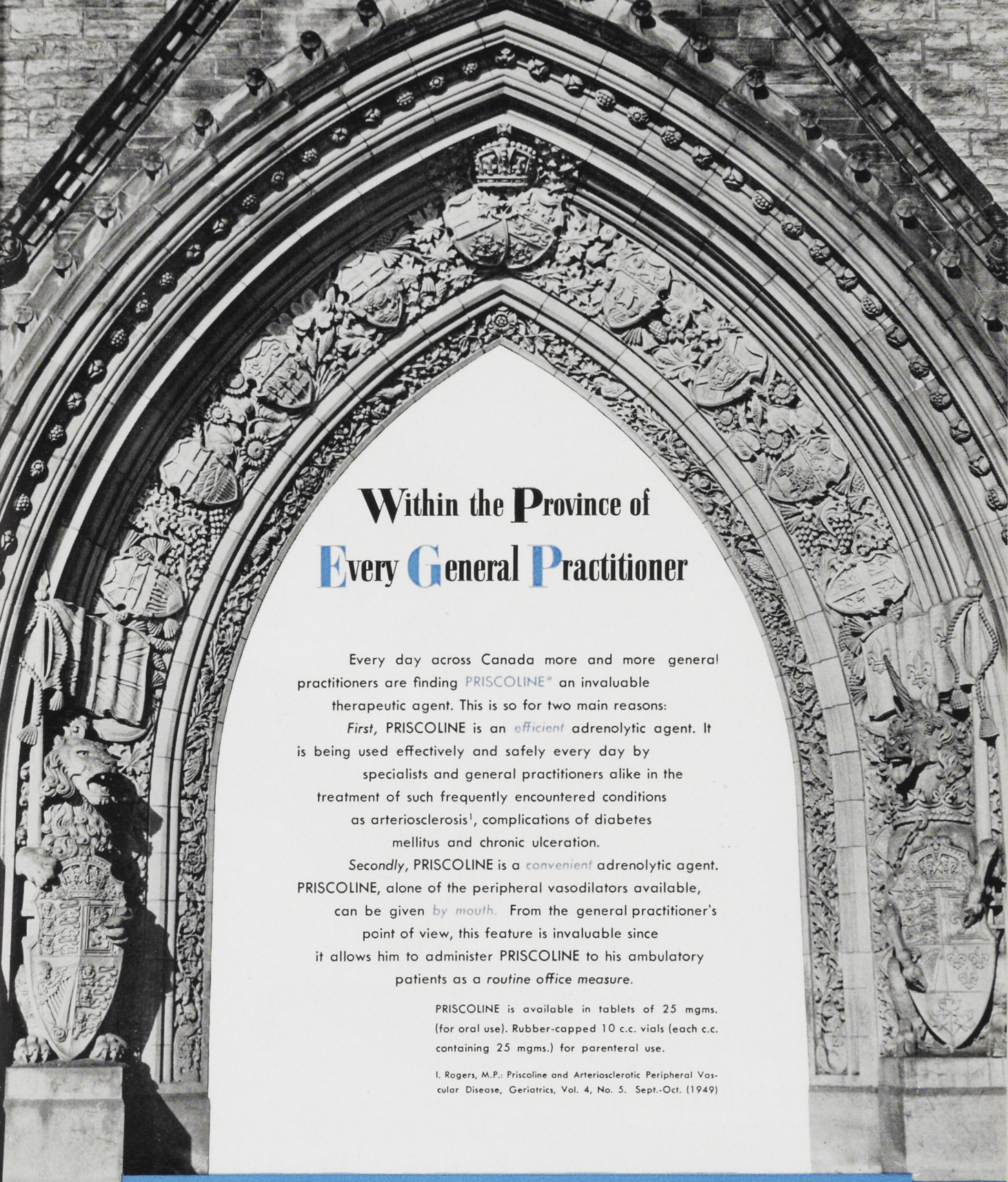
The Physician's Business Practical and Economic Aspects of Medicine, by George D. Wolfe, M.D., Assistant Clinical Professor Otolaryngology, New York Medical College; Fellow, American Medical Association. Foreword by Harold Rypins, A.B., M.D., F.A.C.P., Third Edition, 96 illustrations, 552 pages. J. B. Lippincott Company, Montreal. Price $12.50.

First Aid Manual. This is the official text book of the British Red Cross Society. It is a compact volume of 300 pages illustrated with 168 figures, and contains a larger amount of information than is usual in such handbooks. It is unnecessary to comment upon the usefulness of such a volume.

British Red Cross Society First Aid Manual, No. 1, by Sir Harold E. Whittingham, K.C.B., C.B., F.R.C.P., and Sir Stanford Cade, K.B.E., C.B., F.R.C.S. Ninth Edition, 171 illustrations, Macmillan Co. of Canada Ltd. Price $0.75 cents.

Comparative Anatomy Laboratory Manual. This manual is designed to serve as a guide in dissection and comparative studies of the systems described. It is intended for the use of students but can be of value to others who have occasion to trace the evolutionary development of organs or structures.

Comparative Anatomy Laboratory Manual, by Lloyd Raymond Gribble, Ph.D., Professor of Zoology, West Virginia University, Morgantown, W.Va., Illustrated. The Blakiston Co., Toronto. Price $3.00.



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1. Rogers, M.P.: Priscoline and Arteriosclerotic Peripheral Vascular Disease, Geriatrics, Vol. 4, No. 5. Sept.-Oct. (1949)

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EDITORIAL

J. C. Hossack, M.D., C.M. (Man.), Editor

Freedom from Noise

Freedom is a word which is on everyone's lips these days. Never before have so many people wanted so much, or so many kinds of, freedom. And now I am urging a crusade for still another—freedom for patients from unnecessary noises in hospital.

Every time I pass the Nurses' Home at St. Boniface and see there written over the door "Ecole de Gardes Malades" I think how appropriate it is. Gardes malades—the guardians of the sick. And I think of the Knights Hospitallers of the Order of St. John of Jerusalem—proud nobles who called their charges "Our lords, the sick" and served them as such.

But in no hospital can our lords the sick count on the blessing of quiet, and nowhere can the guardians of the patients shield them against harmful noises. Visitors can, in a measure, be controlled but morning, noon and night in every ward radios blare, bleat, shriek or howl to the infinite distress of all who need peace and quiet for their comfort or recovery.

Noise is a pathogenic agent as surely as any germ. It is as out of place in a hospital as an unsterile instrument, and for that reason should not be tolerated. It irritates, annoys and distresses and so retards improvement. The needs of the most sick, not the comfort of the most convalescent, should be the chief consideration in a ward, and when the welfare of the ill is concerned the mere pleasure of the nearly well must be ignored.

The radio can be a useful means of encouraging convalescence, and if the machine does no more than whisper into its owner's ear it does no harm. But people are thoughtless and common experience shows that patients almost well cannot be trusted to consider the needs or desires of those who are far from well. So we ourselves must see to it that our lords the sick are served as they should be served and guarded as they should be guarded. The freedom to make noise should be taken away from those who abuse it and freedom from noise should be the right of every man and woman who, distressed in body or in mind, seeks rest and comfort in hospital.

There is now in use a radio of which the speaker can be placed under the pillow. Thus can an invalid enjoy his programme while his wardmates enjoy their quiet. The daily fee required is too high for its general use, but at a smaller charge it could go far towards removing a pernicious nuisance. In the meantime we suggest that patients with radios be instructed that a noisy radio will mean no radio at all. It is not fair to the sick that they should have unnecessary and controllable discomforts added to those imposed upon them by disease. Hospital quiet zones should include the hospitals.

* * *

Ten Reviews in 1950

This plump issue is partly a reminder that the current volume will be of ten issues, not twelve as hitherto. Those for June and July will form one and those for August and September will be another.

We should like those concerned to bear this in mind. The tentative programme for the Convention should be in our hands no later than the end of May so that it may appear in the June-July issue to be published on June 15th; and the complete programme should be ready for publication in the August-September issue which will be distributed August 20th.

* * *

The Convention

Concerning the Convention we feel that its success would be assured by the introduction of additional outside speakers. If we could have a Gordon Bell Lecturer, a representative from the American Academy of General Practice, the usual three chosen by the C.M.A. and one or two selected by the Association, large audiences would be attracted. The exhibitors are generous when they are sure of many visitors and more would show their wares if the attendance was certain to be great. If we in Winnipeg can put on the largest bonspiel and the largest musical festival in the world surely we can also put on a very large and attractive Convention, and so long as we must have Conventions let them be the best possible.

General Practitioners of Manitoba

There are about one hundred General Practitioners in Manitoba who have not as yet joined our Association. Believing that there is Strength in Numbers, we solicit your membership in order that we as a group, may successfully carry to a conclusion, a programme that will be beneficial to all General Practitioners.

Enrollment Fee is $5.00. Address your remittance to Dr. A. A. Keenberg, Treasurer, General Practitioners, Association of Manitoba, 901 Boyd Building, Winnipeg.

SOCIAL NEWS

Reported by K. Borthwick-Leslie, M.D.

Sincerest of congratulations to Dr. Elinor Black who has unanimously been elected a Fellow of the Royal College of Obstetricians and Gynecologists, London, Eng. In 1938 Dr. Black successfully wrote the examinations to become a member and for some years was the only Canadian woman member, as she is now the first of our "Fellows." In 1948 Dr. Black was made a fellow of the Royal College of Surgeons of Canada.

♦

Dr. F. W. Jackson, formerly Deputy Minister of Health for Manitoba, and now Director of Health Insurance Studies in the Federal Department of National Health and Welfare, has been awarded the 1950 Gold Medal for "the most outstanding contribution to national or world well being." The presentation was made in Ottawa by President G. E. B. Sinclair of the Professional Institute of Civil Service of Canada.

♦

Dr. Donald Cameron McEwen, of Regina, graduate of the U. of M., has been selected as the first Allain Blair Memorial Fellow of the Canadian Cancer Society.

♦

In a different field, the boys in the north make history. Dr. M. K. Brandt was one of the first to use the new road, travelling by snowmobile from The Pas to Norway House to assist Dr. C. Corrigan perform an operation. Sounds rugged!

♦

The wedding of Mary Elizabeth, daughter of Dr. and Mrs. A. G. Meindl, to Edward Wm. Macfarlane, took place in Winnipeg March 25th. The wedding reception was held at Dr. and Mrs. Meindl's residence. Mr. and Mrs. Macfarlane, following their wedding trip to Minneapolis, will reside in Winnipeg.

♦

Dr. and Mrs. Quentin Jacks have returned very rested, healthy and tanned, from motoring to St. Louis, where Quentin attended the General Practitioners' Annual Convention. He reports a wonderfully well attended — more than 4,000 — organized and conducted meetings. Other Canadians registered were Drs. Morrison and George Black, Vancouver; Goldie Gray, Sarnia; Vic Johnson, Lucknow, and Henry Dunham, Secretary of Ontario Medical. After the meeting the Jacks motored to St. Petersburg, Fla., having a grand holiday at Pass - a Grille. Comments on "Georgia Peaches," I presume to be, re the edible type.

♦

The marriage of Flora Ellen Elsey and Dr. Donald Leslie Wright took place March 11th in St. George's Anglican Church. Following the reception at the Royal Alexandra Hotel, Dr. and Mrs. Wright left for Minneapolis. They will reside in Dryden, Ont.

Dr. and Mrs. Maxwell Rady, have returned from an extensive holiday at Tower Isle, Jamaica, B.W.I.

♦

Drs. Catherine and Jean Whittier, United Church Mission Doctors in Central India, have been guests of Dr. Merle Patterson, who is also at home on furlough from India.

♦

Dr. G. W. Ritchie, U. of M./48 has been appointed Medical Superintendent of our new Princess Elizabeth Hospital. Dr. Ritchie has been on the staff of the Municipal Hospitals for three years, doing special work in Tuberculosis.

♦

Dr. and Mrs. George Sisler have gone to Louisville, Kentucky, where Dr. Sisler will do post-graduate work for the next year.

♦

Congratulations to the parents and welcome to our new juniors:

Dr. and Mrs. F. W. Hayter, a son, Robert Charles, March 11th.

Dr. and Mrs. Gilbert Wheeler, Portland, N.D. (nee Betty Ann Peterkin), on March 4th, a daughter.

Dr. and Mrs. L. W. B. Card (nee Mary Peacock), Nanaimo, B.C., a daughter, Cheryl Ann, March 4th.

♦

Dr. and Mrs. E. W. Pickard, Wildwood Park, Fort Garry, a daughter, Joan Frances, March 23rd.

Merkeley enquires as to where are the "male genes."

♦

Dr. and Mrs. M. R. MacCharles have arrived by air in London, England, and are enjoying their stay at the Mayfair Hotel.

♦

ATTENTION ! — The General Practitioners' Association will hold their Annual Dinner and Dance at the Fort Garry Hotel, Saturday, May 13th. The only "unlucky" omen will be, not to remember the date and arrange your party early.

♦

The Anaesthetists' Banquet on March 23 - 25 was a most enjoyable function. Unfortunately, I was unable to attend the Scientific Sessions, but rumor reports that they were most successful and interesting.

♦

No wonder the Tribune picked "Tony Gowron's new sun deck home on Roslyn Crescent for its excellent article of "Homes Then and Now," Tony and Mrs. Gowron have combined the most satisfactory mixture of modernness, smartness and comfort that I have seen. I love it, and if I didn't live in Fort Garry, would be envious. Oh! alright! I am jealous of that amusement room.

♦

Dr. and Mrs. Norman Corne are also escaping our friendly(?) stimulating, Manitoba weather by holidaying at Miami Beach, Florida.

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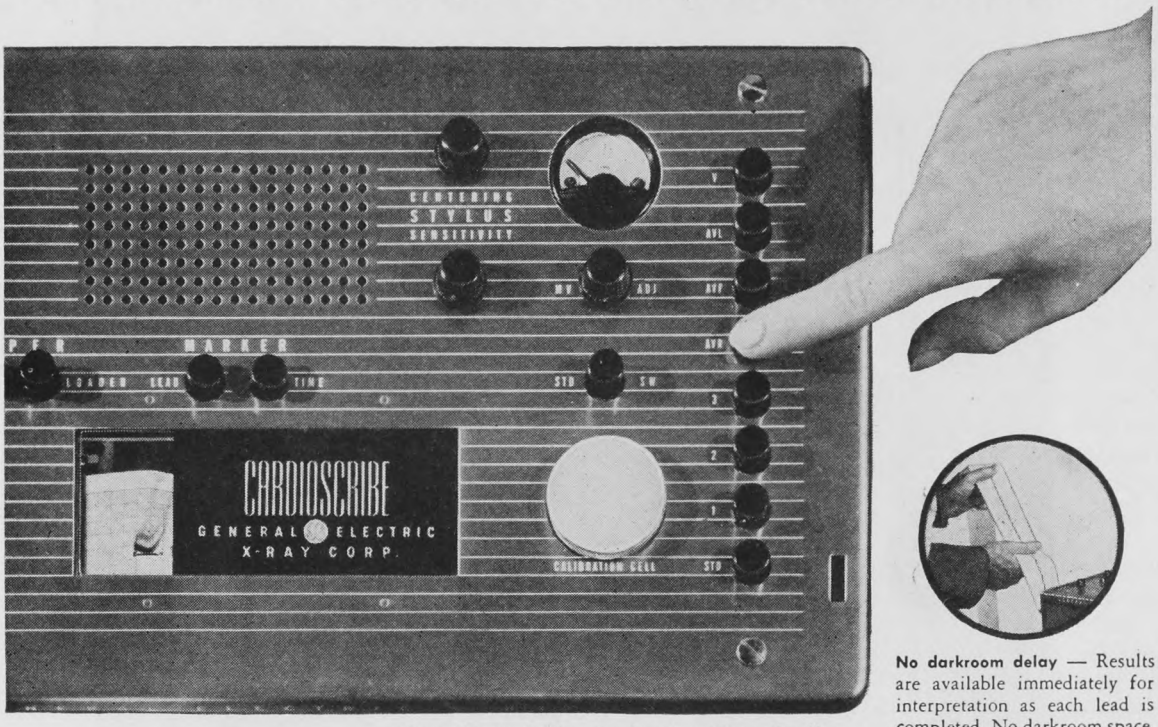

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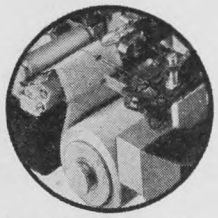

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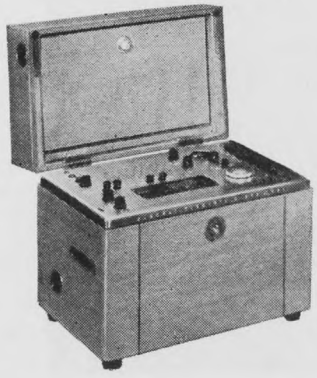

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ASSOCIATION PAGE

Reported by M. T. Macfarland, M.D.

Income Tax

A letter, dated Feb. 28th, 1950, addressed to the Secretaries of Divisions has been received from Dr. A. D. Kelly, Assistant Secretary, Canadian Medical Association:

"Further to my recent letter (Jan. 20th) on income tax returns by members of the medical profession, another aspect of the system of Capital Cost Allowance, applicable to 1949 only, has recently been drawn to my attention.

The law states that, in respect of property on hand at January 1, 1949, its capital cost is reckoned to be its undepreciated value at that date. Any proceeds of disposition in excess of that value is a capital gain and therefore non-taxable. On the other hand, should the proceeds of disposition be less than the undepreciated value at January 1, 1949, the resultant loss is deductible from income or added to the cost of the replacing asset.

For example: Dr. Jones has a motor car which prior to 1949 had been depreciated to a value of $1,500. He disposes of this car to his dealer who allows him $1,800 on the purchase of a new car at $2,500. The $300 is capital gain and need not, in 1949, be deducted from the value of the new asset. He may apply the appropriate capital cost allowance (30%) on the full $2,500 price of his new car.

Conversely, if he should receive a trade in value of only $1,300 on his old car, he has sustained a loss of $200 and this amount may be added to the cost of the new car, and he would begin depreciating it in 1949 at 30% of $2,700.

This information may be of some interest to doctors who during 1949 disposed of assets which are subject to Capital Cost Allowance, and you may feel free to circulate it as you see fit."

Health Survey Committee

The first meeting of the Health Survey Committee set up in this province under the chairmanship of Hon. Ivan Schultz was held at the Marlborough Hotel on Friday, February 24th, 1950. The Committee is composed of laymen and representatives of several professional groups. The Manitoba Medical Association is represented by Doctors E. D. Hudson, A. W. Hogg and R. W. Richardson. Funds for the Committee's work, amounting to $38,000.00, are provided by the Federal Health Grants authorized two years ago to give a national picture of health services and needs. Committee members will advise on the form of the survey, but full-time specialists in the health field will do much of the investigating and reporting. Two public health experts, who made previous surveys in Manitoba, are expected to re-examine public health and make a new report. They are Dr. Carl Buck of the American Public Health Association, and Graham L. Davis of the W. K. Kellogg foundation. Dr. Buck's 1941 survey in Manitoba led to the provincial Health Services Act in 1945. Mr. Davis studied Manitoba hospitals in 1941. While the qualifications of these gentlemen are not questioned, the fact that it is considered necessary to appeal to organizations outside our own country should cause concern.

North of 53 District Medical Society

A meeting of the North of 53 District Medical Society was held at Flin Flon on Wednesday, March 8th, 1950. The meeting was convened by the President, Dr. A. E. McGregor, Sherridon, but the Secretary, Dr. J. M. Ridge, Clearwater Hospital, was unable to be present. Others attending were: Doctors M. K. Brandt and C. S. Crawford, The Pas; Drs. P. Johnson, N. S. Stephansson, H. L. McNicol, G. N. Willson, E. L. Redpath, C. A. Milanese, Flin Flon, and Drs. R. O. Burrell, H. Morison and M. T. Macfarland, Winnipeg.

Following dinner, which was served at the home of Dr. and Mrs. Percy Johnson, the business and scientific sessions were held at the Clinic Office.

The officers were re-elected for the ensuing year by acclamation, and Dr. C. S. Crawford was named representative to the Executive Committee of the Manitoba Medical Association.

Dr. H. Morison spoke on "X-ray Investigation of the Gastro-intestinal Tract," illustrated by films, and Dr. R. O. Burrell conducted a question-answer period on surgical problems, including chronic varicose ulcer, Hirschsprung Disease, congenital megacolon.

A spirited debate on the subject of Manitoba Medical Service and extension to include rural areas culminated in a resolution of approval within certain restrictions.

Applications for Affiliation

The Winnipeg Orthopaedic Society was welcomed as an affiliated group by the Executive Committee on January 15th, while the Eye, Ear, Nose and Throat Section of the Winnipeg Medical Society was similarly welcomed on February 19th.

Searchlights

Representation has been made through the Commissioner of Taxation, the Attorney-General and the Legislative of the Province of Manitoba for an amendment to the Highway Traffic Act which will permit doctors who ordinarily use their cars for night calls to carry searchlights. It is con-

tended that it is not in the public interest to restrict such use since delay in reaching the scene of accident or illness by the doctor may militate against the survival or recovery of the patient.

Blood Transfusion Service

Red Cross Blood Transfusion Service—Dr. C. W. Clark was named as Association representative to the Provincial Committee of the Blood Donor Service, a meeting of which was held on January 5th at the new Red Cross Centre, Osborne St. Official opening of the Centre was held about mid-January, and the first donor clinic was held one week later. Attention is drawn to Dr. Harris' article which appeared on page 89 of the February Review under the caption "Free Blood." One paragraph reads, "the medical and nursing profession should realize their responsibility in this connection. As leaders of thought and action they should be among the first to enroll as volunteer donors." It is understandable that the amount of blood used during the first month was nearly double that of the previous monthly average, indicating that there was a real need for the service, also the fact that what is "free" will be in greater demand. A plea for rational use of the service as a whole, as also for specific types, has already been made by the medical director who is confident that he may have the co-operation of the medical fraternity.

Public Relations—Drs. F. G. Allison and L. A. Sigurdson were named a Committee on Public Relations, with power to add to their numbers. A portion of their activities will be concerned with news releases which will be given to the public, and efforts to acquaint the profession with what is transpiring in the field of medical economics. One of the first duties which will devolve upon the Committee will be co-operation with the Promotional Committee looking towards extension of Manitoba Medical Service to the areas of Manitoba outside of Greater Winnipeg. A series of letters have been prepared by M.M.S. to refresh the minds of city practitioners of the aims and objects of this profession-sponsored experiment for providing prepaid medical care, and to acquaint members of the profession outside Winnipeg of the history and aspirations of Manitoba Medical Service.

General Practice

Resolution of Section of General Practice—At the Annual Meeting of the Canadian Medical Association in Saskatoon, June, 1949, the following resolution was approved, and forwarded by the Section of General Practice to the Executive Committee of the Canadian Medical Association: "That this Section disapproves of the principle of using only Specialty or Fellowship certificates as a criterion of acceptance on hospital staffs as this discriminates against many competent general practitioners." This resolution was accepted and confirmed by the C.M.A. Executive Committee as follows: "1. That consideration be given to the establishment of a Section of General Practice in all general hospitals in which specialists represent a portion of the medical staff. 2. That the criterion by which to judge whether a physician may be a member of the hospital staff should not depend upon certification by the various specialty boards." Each Division was urged that this matter be taken up with hospital boards, and in this province the resolution was referred for study and report to the General Practitioners' Association.

Medical Services

"Medical Services (Canada)" — Delegates from each of the plans for the provision of medical care on a prepaid basis met for two days prior to the last meeting of the Executive Committee of the Canadian Medical Association. The necessity of obtaining a national charter by incorporation was recognized, and a small sub-committee was set up to accomplish this. A resolution was also approved requesting provincial plans to affiliate with the national plan and to make arrangements for looking after a policy-holder regardless of where he resided at the time the service was required. Some system of paying for the service would have to be worked out between the plans. It was pointed out that medical members of existing plans had already contributed a great deal financially to start the prepaid medical care experiment, and that the cost of expansion should be borne by all medical men in Canada, especially since extension of voluntary, non-profit plans is now Canadian Medical Association policy.

Manitoba Society for Crippled Children

A meeting convened by the Council of Social Agencies of Greater Winnipeg met on January 30th to form the Manitoba Society for Crippled Children, a provincial branch of the National Society of Crippled Children, an organization now in its 28th year of operation in Canada and the United States, and with 2,000 affiliates, of which the Ontario Society for Crippled Children is one.

The three-point programme adopted by the Society is:

1. Education of the public, professional workers and parents of handicapped children.

2. Research to provide increased knowledge of the cause of handicapping conditions and the prevention, care and treatment of those conditions.

3. Direct services to the handicapped, including case findings, diagnostic clinics, medical care, physical therapy, occupational therapy, speech and hearing therapy, treatment and training centres and clinics. Special schools and classes, homebound teaching, psychological services, vocational

training, camps, recreational services, social services and provision of braces and special equipment will be part of the programme.

The principal immediate function will be to aid in the 1950 "Easter Seal" campaign, first undertaken by the Kinsmen Club of Winnipeg last year in their work of promoting treatment for spastic children at the Children's Hospital. Mr. Justice A. M. Campbell is the president of the new organization, and the Manitoba Medical Association has been asked to name a representative to the Board of Directors.

"Should We Have National Health Insurance?"

Was the topic of a delayed broadcast on Citizens' Forum on January 12th, in which Dr. Carman C. White, Chairman, C.M.A. Committee on Economics, participated. Also included were a businessman and a free lance writer, and the debate, which was presented in London, Ontario, evoked several questions from the audience. It was subsequently reported in one of the Winnipeg newspapers that the same topic had been discussed by a group in a south-western village of our own province. Few subjects evoke so many well and poorly considered arguments as do those which involve the extent to which social security should be provided for all citizens by a paternalistic government. One factor remains constant in that no such service is or can ever be "free"—someone pays!

Workmen's Compensation Board

It has been brought to the attention of the Executive Committee that while the Workmen's Compensation Board Act and General Information contained in the Surgical Fee schedule indicate that "the injured workman is entitled to have the services of the doctor of his choice, and may not change doctors without the authority of the Board" the employer or employee are not always aware of their rights in this connection, and it may happen that the injured workman is directed to a specific doctor who may be chosen by the foreman on the job rather than the doctor of his choice.

City of Winnipeg New Zoning By-law

The following information is made available through the co-operation of the Medical Health Officer of the City of Winnipeg and his assistants:

"After April 1st, 1950, the New Zoning By-law will take effect. The new by-law will be more restrictive than the old. The principal offices of a physician, dentist, or other person authorized by law to practice medicine or healing will be permitted in an R-1 (One Family District) only if the lot abutts a lot in a less restricted district or is separated therefrom by a lane, and provided that such use is conducted within a one-family dwelling and the residential character of such dwelling is not changed. Doctors' name plates will be permitted in R-1 districts as follows: One unlighted name plate for each dwelling unit, not exceeding one and one-half square feet in area.

The principal offices of doctors will be permitted in R-3 Multiple Family Districts, provided such use is conducted within a one-family dwelling and the residential character of such dwelling is not changed. Permitted name plates will be the same as in R-1 Districts."

City of Winnipeg Diagnostic Unit

Last year a sub-committee of the Health Committee studied and reported to City Council on the possibility of having a Diagnostic Unit set up for the City in conjunction with the Provincial Government under the terms of the Health Services Act, 1945. With a new Council the matter was reopened and a committee was appointed to investigate the cost of setting up such a Diagnostic Unit. The report of the study should be very interesting.

Sixth International Congress of Paediatrics in Zurich

Word was received in January, 1949 concerning the Sixth International Congress of Paediatricians to be held at Zurich, Switzerland, in July, 1950. Subsequently, it was learned that if any member of the profession planned to attend the Congress, at his or her own expense, the Provincial Government would be pleased to designate such a person as official representative. The matter was referred to the Paediatric Section of the Winnipeg Medical Society but at the time of writing no nominee has come forward.

International List of Causes of Death

The following letter, dated January 27th, was addressed to each doctor in the province over the signature of L. E. Stewart, Acting Recorder, Division of Statistics, Department of Health and Public Welfare. For the benefit of those who may not have received the letter it is reproduced herewith:

"The International List of Causes of Death has been revised, and the Sixth Revision was unanimously adopted in April, 1948, by some 29 countries, including Canada and subsequently approved by the World Health Organization and the World Health Assembly for international use. This new list is to replace the 1938 Revision in classification and compiling Canadian mortality statistics as **from January, 1950.**

The problem of classifying causes of death for vital statistics is relatively simple when only one cause is shown on medical certificate of death. However, in many cases two or more morbid conditions contribute to death. In such cases it has been the traditional practice in vital statistics to select one

of these causes for tabulation according to certain rules of preference, and it is mainly in the method of such selection that the new list differs from past revisions.

The selected cause has been variously described in the past as "the cause of death," "the primary cause of death," "the principal cause," etc. In order to make uniform the terminology and procedure for selecting the cause of death for statistical tabulations it was agreed by the International Revision Conference that the cause to be tabulated should be designated the **underlying cause of death.**

The standard medical certificate of death places the responsibility for indicating the train of events on the certifying physician. It is assumed, and rightly so, that the certifying practitioner is in a better position than any other individual to decide which of the morbid conditions led directly to death.

A new edition of the Physicians' Pocket Reference is being prepared and distribution to all practising physicians in Canada will be made within the next few months. It is hoped that this reference book will be of assistance to medical practitioners in certifying causes of death in accordance with this new principle of classification.

May we take this opportunity of expressing our thanks to you for the services and co-operation given this office in the past.

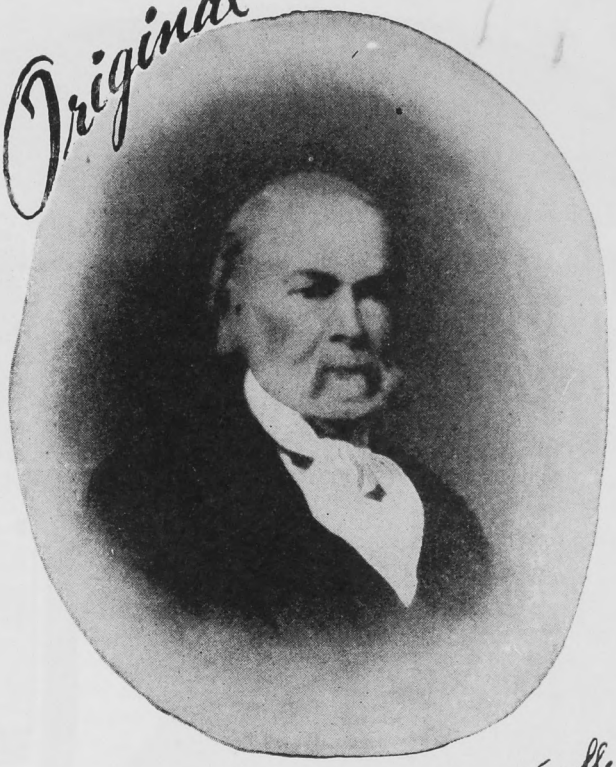
March of Dimes

One of our members complained that the favourite radio programmes to which he was listening were interrupted by the appeal of the National Foundation for Poliomyelitis. Information has increased in the local press, radio, on stamp franks, etc.! This appeal is one which will commend itself to the generosity of many persons since it has the endorsation of Mrs. F. D. Roosevelt, whose late husband had been a sufferer from Poliomyelitis. The lay Board which heads the organization is supported by a medical advisory committee, which includes prominent members of the profession across Canada.

Canadian Arthritis and Rheumatism Society

Mr. James Gairdner, National President of the Canadian Arthritis and Rheumatism Society arranged a luncheon meeting at the Fort Garry Hotel, when Mr. Ralph Baker accepted the Chairmanship of the Manitoba Committee and Mr. Harold Hanson was appointed Treasurer. Dr. J. D. Adamson remains as Chairman of the Medical Advisory Committee. The campaign for funds will commence in May, in anticipation of which Mr. Baker is expected to speak to the Winnipeg Medical Society.

Original



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COLLEGE OF PHYSICIANS AND SURGEONS OF MANITOBA

Council Meeting

Winnipeg, Manitoba, October 19, 1949

(Continued From March, 1950, Issue)

5. Reports of Special Committees and their consideration.

(a) **Representatives to the Manitoba Medical Association Executive**

Dr. Edward Johnson stated he had nothing to report.

Request for Grant for Extra Mural Post Graduate Work

(Refer Finance Committee 4 (d)).

(b) **Trustees of the Gordon Bell Memorial Fund**

No report.

(c) **Representatives to the Committee of Fifteen**

No report.

(d) **Representative to the Committee on Admissions**

Your representative on the student selection committee for entry to first year medicine being out of town I attended in his stead as arranged at our May meeting. Dr. Chown, in report a year ago, stated he was dissatisfied with the way selection is arrived at and in this I am in agreement with him. There were well over 200 applicants being 3 for every possible entrant. Among them were 62 graduates in Arts and Sciences.

No student arriving at the time of entry to Medical College is unable to get sponsors who will write letters of commendation and there is no method to assess these so that they mean nothing. Personal interviews by the Dean have become impossible with so great a number of applicants. So that selection has come to be a matter of academic averages and the fulfilling of standard subject requirements. Veterans preference has now taken care of most of the student veterans of satisfactory ability and is no longer a factor of any moment. Veteran applicants receiving the benefit of preference if their standing was nearly commensurate with that required of others.

The only yardstick that could be applied was the fulfillment of required subjects and a high overall average. Those students or graduates of Manitoba University having over 75% average and all required subjects were first considered and listed, then those over 70%, then those over 67½%. Only one student from outside the province was admitted and he because he had formerly fulfilled the requirements here and been accepted and has received permission to delay his entry for two years' study at McGill.

I think we ought all to give some thought to the method of selection and it seems to me that examination marks alone should not entirely determine acceptance. Experience has proven that the student who can make the highest marks does not usually make the best physician.

I should like to see acceptance determined by a percentage system with 75% for overall examination average and the balance divided according to the students extra curricular activities and personality appraisement by capable interviewers. This would allow the committee on admission to have the benefit of an appraisal of the applicants personality which is impossible when only names, not necessarily the original ones, and yearly examination average are available. The applicant for all I know may be a stuttering, cross-eyed, misfit.

There is general agreement that the present selection method is unsatisfactory and does not select those of the applicants who will be likely to contribute most to the health of the population and the advancement of the art and science of healing.

Respectfully submitted,

T. H. Williams, M.D., C.M.,
D.T.M. & H., F.C.A.P.

Motion: "THAT the report of the Representative to the Committee on Admissions be adopted." Carried.

Dr. Corrigan stated that the method of selection has been laid down by the University, and perhaps by higher authority to them, and inquired whether it would be possible for this Council to make a change.

Dr. Williams considered the Council should make a recommendation, if everyone just sits back, nothing will be done about it.

Motion: "THAT the Council of the College of Physicians and Surgeons of Manitoba suggest that in acceptance of medical students to the Faculty of Medicine, it be necessary that there be an interview and a satisfactory personality evaluation." Carried.

(e) **Representatives to the Medical Council of Canada**

The Medical Council of Canada met in Ottawa for a two-day session, 14th and 15th September. Several members of Council were absent. These included my colleague, Dr. Brian Best, whose absence was much deplored.

The report of the Registrar showed that there were now 10,751 Licentiates of the Council—581 having been granted this year—all but two by examination. The assets of the Council were placed at $145,000.

The committee on the issuance of Enabling Certificates had done nothing and asked to be continued another year.

The discussion in regard to Licentiate examina-

tions elucidated that there were three methods in use.

1. Dalhousie University has its written examinations at the end of the 4th year, its orals and graduation at the end of the 5th year.

2. Toronto gives the full examination and graduation at the end of the 4th year. The hospital interneship then follows and the internes are all graduates with no examination prospects to worry them. The Ontario College of Physicians and Surgeons Registrar was not very definite about allowing these men to practice without the interne year.

3. Manitoba gives the full examination at the end of the 5th year.

Newfoundland sent word that they would be pleased to join the Medical Council of Canada, and a motion was introduced to allow all Newfoundlanders who had been in practice 20 years before the entrance of their country into Confederation, to become Licentiates of the Council without examination. However, it was pointed out that this would be contrary to the Medical Act and apart from this was inadvisable. The different Canadian universities appear agreed on the advisability of coincidental examinations, but in some cases there are difficulties. For instance the C.P. & S. in Quebec insist that they shall have representatives taking active part in all medical examinations.

The main body of examiners points out that some of the oral examiners are very lenient. A candidate may make 45 or 50% in his written surgery and 98% or over in the oral.

The two-day session was very satisfactory and cleared the slate of practically everything but the Enabling Certificate and the foreign graduates. These are still in the lap of the provincial bodies.

Dr. Bramley-Moore submitted a pamphlet outlining a scheme to protect the public from medical men who attempt procedures for which they are not qualified. In his scheme, on graduation, a man would be granted a basic certificate allowing him to do minor surgery, non-surgical obstetrics and the closed treatment of fractures. After a certain time passed in general practice he might, after further study and examination, be given a specialits's degree. The effect of this plan, if followed, would be to prevent the general practitioner doing work that requires a specialist. This matter was referred to the various provincial bodies.

Respectfully submitted,

J. S. Poole, M.D., C.M.

Motion: "THAT the report of the Representatives to the Medical Council of Canada be adopted." Carried.

(f) **Representative to the University Senate**

The Registrar read the following report:

As your representative to the Senate of the University of Manitoba I beg to report that since the last Annual Meeting of the Council I have attended all but one of the regular monthly meetings of the Senate.

In addition, I have served on the following standing committees of the Senate: The Committee on Examinations for Student Nurses, the Advisory Committee on Nursing Education, and the Committee on Administration of the Basic Sciences Act.

During this period Certificates of Credit in the Basic Sciences have been issued to 76 medical graduates, distributed as follows: 36 Manitoba, 15 other provinces of Canada, 13 Great Britain, 6 U.S.A., 2 continental Europe, 3 China, 1 Australia. One non-medical graduate of the Canadian Memorial Chiropractic College was issued a Certificate of Credit.

The Committee has recommended that the fee for the Basic Sciences Certificate should be increased from $1.00 to $5.00.

All of which is respectfully submitted,

J. M. LEDERMAN, M.D.

Motion: "THAT the report of the Representative to the University Senate be adopted." Carried.

(f) **Representatives to the Cancer Institute**

Dr. Macfarland reported that the Registrar and President are members exofficio of the Board of the Cancer Relief and Research Institute.

It was reported in May that approach had been made to the problem of providing Cancer Diagnosis Clinics for the residents of rural Manitoba. Although the federal grant was allotted for cancer control in the whole province, such grant was a matching grant and dependent on the amount of money the province was prepared to devote to the disease.

When the initial plan was prepared there were controversial points raised, and not only did the executive of the Manitoba Medical Association request reconsideration, but the plan was referred back to the Institute by the hospitals, suggesting that the proposed plan, or an alternative plan which was acceptable to the respective medical staffs and the medical profession as a whole, would be satisfactory to the hospital management. At the Annual Meeting of the Manitoba Medical Association, a resolution was adopted to the effect that the so-called operational plan was unacceptable, and the matter be referred to the new Executive Committee with power to negotiate an alternative agreement with the Institute. (Such an alternative was accepted in principle by the Association Executive on October 13th subject to submission of detailed plan by the Cancer Relief and Research Institute to the Association Executive for ratification).

Motion: "THAT the report of the Representatives to be Cancer Institute be adopted." Carried.

(h) **Representatives to the Liaison Committee—M.M.A. & C.P. & S.**

The Registrar reported that on the advice of the Liaison Committee a Mail-A-Voice recording machine had been purchased at a cost of $378.67. The C.P. & S. share in this machine would be $189.33.

Motion: "THAT the C.P. & S. pay to the M.M.A. the sum of One Hundred and Eighty-nine Dollars and Thirty-three Cents ($189.33), being one-half share in the Mail-A-Voice machine." Carried.

6. Election of Officers and Standing Committees.

Officers:

(a) **President**

Motion: "THAT Dr. Edward Johnson be appointed President." Carried.

(b) **Vice-President**

Motion: "THAT Dr. I. Pearlman be appointed Vice-President." Carried.

(c) **Registrar**

Motion: "THAT Dr. M. T. Macfarland be appointed Registrar." Carried.

(d) **Treasurer**

Motion: "THAT Dr. T. H. Williams be appointed Treasurer." Carried.

Nomination Committee to Strike Standing Committees

The following members were appointed from the Chair, to be a committee to strike Standing Committees:

Dr. B. D. Best, Dr. Edward Johnson and Dr. A. A. Alford.

Dr. C. B. Stewart vacated the Chair in favour of the newly elected President, Dr. Edward Johnson.

Standing Committees

(a) **Registration Committee**—Dr. C. H. A. Walton, Chairman; Dr. I. Pearlman, Dr. C. E. Corrigan.

(b) **Education Committee**—Dr. A. A. Alford, Chairman; Dr. A. L. Paine, Dr. W. J. Boyd.

(c) **Finance Commitee** — Dr. T. H. Williams, Chairman; Dr. C. S. Crawford, Dr. B. Dyma.

(d) **Legislative Committee** — Dr. J. S. Poole, Chairman; Dr. B. D. Best, Dr. F. K. Purdie, Dr. T. W. Shaw, Dr. C. W. Wiebe.

(e) **Discipline Committee** — Dr. A. A. Alford, Chairman; Dr. C. W. Wiebe, Dr. C. E. Corrigan, Dr. C. B. Stewart, Dr. H. Guyot.

(f) **Executive Committee**—Dr. C. B. Stewart, Chairman; Dr. J. S. Poole, Dr. I. Pearlman, Dr. C. H. A. Walton, Dr. B. D. Best.

(g) **Library Committee**—Dr. B. D. Best.

(h) **Taxing Committee**—Dr. C. W. Wiebe, Chairman; Dr. B. Dyma, Dr. C. B. Stewart.

Motion: "THAT the appointment of Standing Committees be accepted." Carried.

Election of Special Committees

(a) **Representatives to the Manitoba Medical Association Executive**

Motion: "THAT our representatives to the Manitoba Medical Association Executive be Dr. C. B. Stewart and Dr.Edward Johnson." Carried.

(b) **Representatives to the Committee of Fifteen**

Motion: "THAT our representatives to the Committee of Fifteen be Dr. B. D. Best, Dr. I. Pearlman and Dr. Edward Johnson." Carried.

(c) **Representative to the Committee on Admissions**

Motion: "THAT our representative to the Committee on Admissions be Dr. T. H. Williams." Carried.

(d) **Representatives to the Medical Council of Canada**

Motion: "THAT our representatives to the Medical Council of Canada be Dr. J. S. Poole and Dr. B. D. Best." (Nominated in 1948 for term of 4 years). Carried.

(e) **Representative to the University Senate**

Motion: "THAT our representative to the University Senate be Dr. C. H. A. Walton." Carried.

(f) **Representatives to the Liaison Committee—M.M.A.-C.P. & S.**

Motion: "THAT our representatives to the Liaison Committee—M.M.A.-C.P. & S. be Dr. B. D. Best, Dr. A. A. Alford and Dr. Edward Johnson." Carried.

Appointment of Auditors

The Registrar explained that the Auditor's charges have increased from $40.00 in 1943 to $75.00 in 1944, $100.00 in 1947 and $125.00 in 1948. He stated that last year he requested an estimate of the possible charges for the year ending September 30, 1949, and received an estimate of $125.00. This year he received an estimate of $150.00 for the ensuing year. He was informed that the 1948 costs were actually $175.00, and the costs for this year were $119.00 which, with secretarial help, would not allow for a fair margin of profit.

Motion: "THAT the appointment of auditors be deferred until the May meeting, and that the Finance Committee make inquiries as to whether or not a change should be made." Carried.

Appointment of Scrutineers

Motion: "THAT Dr. Flinor Black and Dr. A. R. Birt be appointed scrutineers, and Dr. D. Swartz and Dr. W. T. Dingle be appointed alternate scrutineers, for the ensuing three-year period." Carried.

7. Reading of Communications, Petitions, etc., to the Council.

(a) **Communication From Registrar, C.P. & S., Alberta, Re Basic Licence**

The Registrar reported that a copy of Dr. Bramley-Moore's notes concerning a Basic Licence had been forwarded to each member of Council. He stated that the matter had been discussed at the Registrars' Meeting and that Dr. Poole had reported that it was discussed at the meeting of the Medical Council of Canada. In each instance

it was referred to the individual provincial councils.

The matter was referred to the Education Committee for study.

(b) **Petition Re Reinstatement of Dr. .....**

A request was received from Dr. ..... for reinstatement on the Register of the C.P. & S., Manitoba. His application was supported by several letters of recommendation.

Motion: "THAT the name of Dr. ..... be reinstated on the Register of the College of Physicians and Surgeons of Manitoba, on payment of the fee of One Hundred Dollars ($100.00)." Carried.

(c) **Communication From C.P. & S., British Columbia, Re Recognition of Specialists**

The following regulations were made by the Council of the College of Physicians and Surgeons of British Columbia re recognition of specialists:

1. As from July 22, 1949, the Council of the College of Physicians and Surgeons of British Columbia will grant recognition in the various specialties only to those who hold:

Certificate or Fellowship in the Royal College of Physicians and Surgeons of Canada.

2. As from July 22, 1949, the council of the College of Physicians and Surgeons of British Columbia may grant **interim recognition** for one year, and renewed, if necessary, up to three years to those men who hold the qualifications of any one of the following:

(a) Fellowship in Surgery of the Royal Colleges of the British Empire.

(b) Membership of the Royal Colleges of Medicine or of Obstetrics and Gynaecology of the British Empire.

(c) The Diploma of an American Board.

and

All interim recognition so granted by this Council shall cease as of the first Monday in May, A.D. 1952, and, thereafter, the Certification or Fellowship of the Royal College of Physicians and Surgeons of Canada shall be the only basis for recognition of specialists in the Province of British Columbia.

Dated this 30th day of July, A.D. 1949.

Dr. ..... stated that the matter of specialist recognition had been a problem to the Manitoba Medical Service. He said that the M.M.S. had asked on many occasions for a specialist register, but were unable to get direction from any body. He stated that the M.M.S., under necessity, had classified specialists, adopting a plan nearly the same as that of B.C. He suggested that it was the duty of the Council to act, instead of avoiding it further.

Dr. ..... advised that the provinces of Quebec, Ontario, Alberta and British Columbia had specialist registers, and said that requests often come to the office for lists of specialists.

Dr. ..... thought the question should be directed to the committee to get the most direct action, and suggested the Legislative Committee.

Dr. ..... said it would be necessary to have recommendations first, before implementation.

Dr. ..... thought it was a very big question, and concerned the University of Manitoba, the Manitoba Medical Association, the Manitoba Medical Service, and also appointments to hospitals. He said the Council should accept standards first, and then implement them.

Motion: "THAT the question of specialist register be referred to the Education Committee for study and recommendation." Carried.

(d) **Communication From Canadian Medical Association, Re Indian Students Obtaining Post Graduate Training in Canada**

The Registrar presented a communication from Dr. A. D. Kelly, Assistant Secretary, Canadian Medical Association, enclosing letter from Dr. S. C. Sen, New Delhi, India. Dr. Sen outlined the need of doctors in India, and stated that they need specially qualified people in all specialties who will not only give adequate medical care to the people of India, but also act as teachers in the new colleges to be started. He inquired whether Canada could help train India's future teachers in the various medical centres. He stated that the Indian Medical Association would make careful selection of young doctors, who would be full university graduates possessing qualifications registrable in the United Kingdom, and send them out for the period of study extending from 2 to 5 years as the case may be.

Motion: "THAT the question of accepting Indian doctors for study in Canada be referred to the Executive Committee." Carried.

(e) **Communication From the Canadian Arthritis and Rheumatism Society, Manitoba Division**

The Registrar presented a letter from the Manitoba Division of the Canadian Arthritis and Rheumatism Society, requesting a representative from the College. He stated that he had unofficially represented the College at two meetings of the Division, held for the purpose of organization.

Motion: "THAT Dr. M. T. Macfarland be appointed our representative to the Canadian Arthritis and Rheumatism Society, Manitoba Division." Carried.

(f) **Communication From Dr. .....**

A request was received from Dr. ....., now practicing in Alberta, for a refund of his registration fee. Dr. ..... took out Temporary Licence on November 24, 1947, when he was employed by the Provincial Government. On November 20, 1948, he took out Permanent Registration and practiced at ..... until the 30th of September, 1949.

Motion: "THAT Dr. .....'s request for refund of registration fee be refused." Carried.

(g) **Communication From the Motor Vehicle Branch**

The Registrar presented a communication from the Deputy Registrar of the Motor Vehicles, advising that the numbering system of licence plates for passenger cars for 1950 will not include a 4,000 series, and that in lieu of that, it may be possible to issue series 4D1 to 4D999 to registered physicians in Manitoba.

Motion: "THAT the Deputy Registrar of Motor Vehicles be requested to set aside licence plate series 4D1 to 4D999 for physicians registered in Manitoba." Carried.

8. Inquiries.

None.

9. Notices of Motion.

None.

10. Motions of which notice has been given at a previous meeting.

None.

11. Unfinished business.

None.

12. Miscellaneous and New Business.

(a) **Payment of Janitor**

Motion: "THAT the janitor be paid Five Dollars ($5.00) for his services at this meeting, plus the charge for refreshments." Carried.

(b) **Amount to be Paid to Council Members for This Meeting**

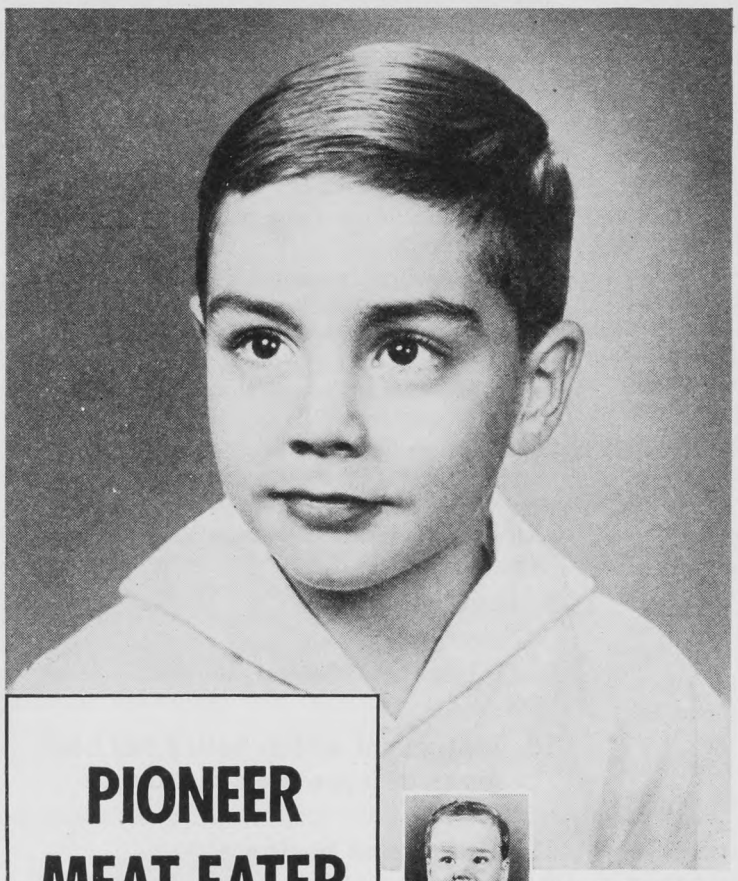
Motion: "THAT the rate of payment to Council members present at this meeting be Twenty-five Dollars ($25.00) for city members, and for rural members, Twenty-five Dollars ($25.00) for each day required to be absent from his practice, and the mileage at the rate of Ten Cents (10c) per mile both ways." Carried.

(c) **Motion Re Salaries and Amount to Be Paid to the Manitoba Medical Association Each Month**

Motion: "THAT the salary of the Registrar be Two Hundred Dollars ($200.00) per month, the Treasurer be Five Hundred Dollars ($500.00) per year, and that the Mantioba Medical Association be paid Two Hundred Dollars ($200.00) per month for office and secretarial expenses." Carried.

Adjournment

Motion: "THAT the meeting be adjourned." Carried.



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IN Swift's original test feedings, Kenny Gorman started Swift's Meats for Babies at *three months*—right along with his first solid foods. Contrast this with the general practice five years ago. Before the development of specially prepared Swift's Meats for Babies, the customary age to start meat was *six* or *nine* months. And because home-preparation was so difficult and tiresome, most babies received very little meat—or simply went without!

Kenny's mother will verify the fact that he's as "big and husky" as he looks in his photograph. Says Mrs. Gorman, "He can hold his own against my other three children."

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REPORT NO. 2

SLEEP CHARACTERISTICS OF THE HUMAN INFANT

An investigation of the sleeping habits of infants six to twenty-six weeks of age is being made in this study. The effect of diet on the sleeping habits of the infants under study is one phase of the investigation. The test diet contains Swift's Strained Meats. Special apparatus attached to the cribs records the movement of the children and indicates their periods of sleeping and wakefulness.

This study is part of an extensive clinical research program now being conducted through grants-in-aid made by Swift's.

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The University of Manitoba, Faculty of Medicine

Recent Accessions, 1948-49

General List

Adair, F. H. Maternal care complications; the principles of management of some serious complications arising during the antepartum and postpartum periods, approved by the American Committee on Maternal Welfare . . . 2nd ed.
Univ. of Chicago Press, 1941. 93 p.

Adler, Alfred. Understanding human nature.
Garden City, 1927. 286 p.

Allen, R. B. Medical education and the changing order.
Commonwealth Fund, 1946. 142 p.

Anderson, D. G. The therapeutic value of penicillin.
Edwards, 1948. 297 p.

Archer, V. W. The osseus system; a handbook of roentgen diagnosis.
Year Book, 1945. 320 p.

Ariëns Kappers, C. U. The comparative anatomy of the nervous system of vertebrates, including man.
Macmillan, 1936. 2 v.

Adler, Alfred. The education of children.
Greenberg, 1930. 309 p.

Autenrieth, Wilhelm. Die auffendung der gifte und starkwirkende arzneistoffe zum gebrauch in chemischen laboratorien. 6 neuarbeitete.
Sternkopff, 1943. 343 p.

Bamford, Frank. Poisons: their isolation and identification.
Churchill, 1947. 304 p.

Barach, A. L. Physiologic therapy in respiratory diseases. 2nd ed.
Lippincott, c1948. 408 p.

Barlow, Peter. Tables of squares, cubes, square roots, cube roots and reciprocals . . . 4th ed.
London, 1947. 258 p.

Department of Health and Public Welfare

Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1949		1948		Total	
	Jan. 29 to Feb. 25,'50	Jan. 1 to Jan. 28,'50	Jan. 30 to Feb. 26,'49	Jan. 2 to Jan. 29,'49	Jan. 1 to Feb. 25,'50	Jan. 2 to Feb. 26,'49
Anterior Poliomyelitis	2	0	0	0	2	0
Chickenpox	170	172	137	160	342	297
Diphtheria	1	2	6	2	3	8
Diphtheria Carriers	0	0	2	0	0	2
Dysentery—Amoebic	0	0	0	0	0	0
Dysentery—Bacillary	5	0	2	0	5	2
Erysipelas	6	3	1	3	9	4
Encephalitis	0	0	0	0	0	0
Influenza	7	2	10	5	9	15
Measles	116	147	711	350	263	1061
Measles—German	1	0	0	5	1	5
Meningococcal Meningitis	3	2	0	1	5	1
Mumps	49	26	152	148	75	300
Ophthalmia Neonatorum	0	0	0	0	0	0
Pneumonia—Lobar	14	4	11	5	18	16
Puerperal Fever	0	0	1	0	0	1
Scarlet Fever	65	28	14	16	93	30
Septic Sore Throat	4	5	4	0	9	4
Smallpox	0	0	0	0	0	0
Tetanus	0	0	0	0	0	0
Trachoma	0	0	0	0	0	0
Tuberculosis	56	39	45	14	95	59
Typhoid Fever	0	0	0	0	0	0
Typhoid Paratyphoid	0	0	0	0	0	0
Typhoid Carriers	1	0	0	0	1	0
Undulant Fever	0	0	2	0	0	2
Whooping Cough	21	5	26	4	26	30
Gonorrhoea	77	97	94	97	174	191
Syphilis	19	24	38	39	43	77
Diarrhoea and Enteritis, under 1 yr.	15	1	8	5	16	13

Four-Week Period January 29th to February 25th, 1950

DISEASES (White Cases Only)	*779,000 Manitoba	*861,000 Saskatchewan	*3,825,000 Ontario	*2,962,000 Minnesota
*Approximate population.				
Anterior Poliomyelitis	2	1	2	....
Diarrhoea and Enteritis	15	2	....	....
Chickenpox	170	154	1275	....
Diphtheria	1	....	8	8
Dysentery—Amoebic	....	....	1	2
Dysentery—Bacillary	5	....	4	2
Encephalitis	....	....	1	....
Erysipelas	6	2	4	....
Infectious Jaundice	....	....	30	....
Influenza	7	6	36	....
Measles	116	261	2380	272
Measles, German	1	151	1095	....
Meningitis Meningococcal	3	....	2	7
Mumps	49	168	2499	....
Pneumonia Lobar	14	....	....	....
Puerperal Fever	....	....	1	....
Scarlet Fever	65	9	185	108
Septic Sore Throat	4	1	10	27
Tuberculosis	56	32	189	31
Typhoid Fever	....	....	2	2
Typh. Para. Typhoid	....	....	1	....
Typhoid Carrier	1	....	....	....
Undulant Fever	....	....	5	24
Whooping Cough	21	39	594	76
Gonorrhoea	77	....	202	....
Syphilis	19	....	133	....

Chickenpox, Measles, Mumps, Scarlet Fever and Tuberculosis are the diseases showing fairly large numbers in this four-week report. None of them, excepting **Scarlet Fever**, are higher than average incidence. The Scarlet Fever continues to be a mild type.

The Typhoid Carrier is a Treaty Indian from the Indian Springs Reserve.

Syphilis continues to show a definite decrease in numbers, also gonorrhoea to a lesser extent.

♦

DEATHS FROM REPORTABLE DISEASES

January Registrations Received Since Feb. 14, 1950, and February Registrations Received to March 14, 1950

Urban—Cancer, 50; Influenza, 1; Pneumonia of newborn, 1; Pneumonia Lobar (108, 107, 109), 3; Pneumonia (other forms), 8; Tuberculosis, 5; Gastro-enteritis, 1; Meningococcal infection, 1; Diarrhoea of newborn, 1; Hodgkin's Disease, 1. Other deaths under 1 year, 19. Other deaths over 1 year, 197. Stillbirths, 20. Total, 236.

Rural—Cancer, 20; Diphtheria, 1; Pneumonia Lobar (108, 107, 109), 2; Pneumonia (other forms), 10; Puerperal Septicaemia, 1; Tuberculosis, 12; Gastro-enteritis, 2; Chickenpox, 1. Other deaths under 1 year, 26. Other deaths over 1 year, 147. Stillbirths, 11. Total, 184.

Indians—Influenza, 2; Pneumonia of newborn, 3; Pneumonia (other forms), 2; Tuberculosis, 4; Gastro-enteritis, 1. Other deaths under 1 year, 2. Other deaths over 1 year, 4. Total, 6.

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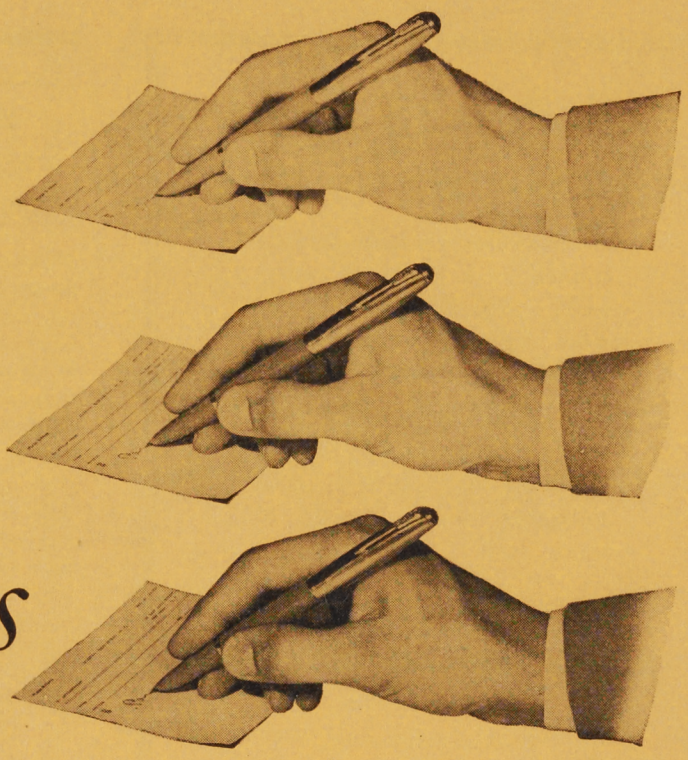

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